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ORAL MUCOUS MEMBRANE MANIFESTATIONS OF SYSTEMIC DISEASE*

ALICE E. PALMER, M.S., M.D.†

Mr. Chairman, members of the American Academy of Dental Medicine and guests:

May I first express my appreciation of the honor you have given me in asking me to address your Society. It is indeed a challenge to speak about oral mucous membrane lesions to such a highly specialized group.

My topic is a broad one. There is a host of systemic diseases which involve the oral mucous membranes. These include allergy, disorders of metabolism, deficiency diseases, congenital, pigmentary, neurological, bacterial, fungal, protozoal, venereal and the systemic neoplastic diseases. You have already covered the material relating to endocrine and hematologic diseases. This presentation is a general summary of this great body of information from the dermatologists' viewpoint.

Now all of you are acquainted with the anatomy of the mucous membrane in the lips and mouth. From a dermatologic point of view, it is important to remember that the mucous membrane is made of stratified squamous epithelium with an underlying loose connective tissue. The mucous membrane is generally loosely attached to the tissues beneath it except over the hard palate. This influences the picture produced by eruptions. However, and this is a most important point, there is no horny layer, or stratum corneum, as exists everywhere on the skin as such. Furthermore, we can readily see that there will be a profound difference in the appearance of lesions in the mouth and those on the skin when we recall that the mucous membranes are constantly bathed by secretions, with varying pH and chemical content. Remember then, that in the systemic disease with eruptive manifestations, the eruptions in the oral mucous membranes are usually identical with those found on the skin proper, but the constant maceration, heat, moisture, friction, and dissolving action of the saliva alter the appearance.

NOMENCLATURE

In dermatology we speak of an *eruption* as being the entire cutaneous manifestation of a disease. A *lesion* is the isolated unit of the eruption, one or more of which make up the eruption. We think of lesions as being either *primary* or *secondary*. A primary lesion is the original phase of the lesion.

A secondary lesion is one which used to be primary but is now changed into something else. The primary lesions are: the macule, the papule, the vesicle, the wheal or hive, and the nodule. These may vary greatly in size, and also in color. The macule is actually a spot, or non-elevated lesion, identified only in that its color is different from the surrounding area. Macules may be small as in scarlatiniform eruptions, or larger, as in measles, or morbilliform eruptions. They may be white, yellow, or any shade of pink, salmon or red, or, they may be purpuric or pigmented. The latter is exemplified by the freckle. If a lesion is localized and has the third dimension of elevation added, and if this elevation is due primarily to actual cellular infiltration, the lesion is called a papule. This condition on a larger scale, with almost more vertical elevation than horizontal, is called a *nodule*. When this process is much exaggerated the lesion is called a *tumor*. The third important primary lesion is the vesicle. This phenomenon represents actual accumulation of fluid and may occur at any level of the epidermis. The fluid may be within the cells themselves, or between the cells. When the vesicle is unusually small it is called a bleb. When it is large it is called a *bulla*. The bulla may represent just a large vesicle or it may result from the confluence of

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many vesicles. The hive, or wheal, represents an accumulation of fluid very deep in the epidermis plus a peculiar vascular response as a halo. Good clinical diagnosis of eruptions depends largely on a vast experience in recognizing the classic as well as the aberrant forms in which these primary lesions arrange themselves, with regard to size, color and distribution.

After the primary lesions have appeared they usually undergo some sort of change. The manifestations of these changes are called secondary lesions. Among these is, for instance, the *scale*. This represents either extra formation of cells in the horny layer, or may just be extra cells formed from over-activity of the epidermis and which are not loose enough to be sloughed off. Another secondary lesion is the pustule, which is really a vesicle into which have seeped various blood elements or bacteria, and the like. On exposure to air this debris dries out and is called a *crust*. When skin becomes thick and dry it may crack and the result is called a *fissure*. Scratches or excoriations are also examples of secondary lesions, as are ulcerations and scars. Now we mentioned earlier that there is no horny layer in the mucous membranes, so there can be no scales. Likewise, oral lesions cannot retain a crust because of the dissolving and macerating effect of the mucosal secretions. Once in a while there will be seen in the mouth a lesion with a pseudomembranous ring around it which is all that remains of what would have been a crust on the skin.

SKIN AND ORAL MUCOUS MEMBRANE

There are some other anatomical differences between the skin and the oral mucous membranes. Among these is the absence of hairs, which automatically eliminates the possibility of a large group of cutaneous diseases. The skin also has a large number of sweat glands, which are lacking in the mouth. This precludes the development of another large group of manifestations in the mouth. However, the mucous membrane has a few distinctive qualities of its own. The presence of mucous glands gives rise to the possibility of diseases affecting them. Also the juncture of mucosa with skin, the mucocutaneous junction, or the vermillion border is subject to special hazards.

There is another major difference between skin and mucous membranes. This difference is related directly to the geography and function of the mucous memberane. In the mouth the mucosa is subjected to appliances, movements, and contacted agents that are usually quite different from those affecting the skin in general. Even the type of bacteria and parasites which thrive in the mouth are selective by reason of the moisture, pH, heat and other factors. There are many lesions on the mucous membrane which, if viewed alone, and out of context with the lesions which may be on the skin, will go undiagnosed, or, more commonly, misdiagnosed. The dermatologist has an advantage in that we routinely examine the nails, hair and skin, as well as the mucous membrane.

Fig. 1. A lesion of chicken pox.

Now, while the skin and mucous membranes exhibit differences, they have some similarities. Both may exhibit inflammations, either acute or subacute, or chronic. They both may show manifestations of allergies, either the localized contact type or the systemic type. The latter may be due to infectious allergy, or to chemical, drug, or botanical allergens. These allergic reactions may be in the nature of urticarial or eczematous manifestations.

Both the skin and the mucous membranes may be damaged by a host of systemic infec-

tions, either acute or subacute, or chronic, including such diseases as:

1. The exanthems, such as measles, scarlet fever, chicken pox, (Fig. I).
2. The collagen diseases, which include scleroderma, periarteritis nodesa, and lupus erythematosus.
3. The infectious granulomatous diseases, such as tuberculosis, syphilis, leprosy, histoplasmosis, leishmaniasis, blastomycosis, and related conditions.
4. The pyogenic infections, such as bacteremia.
5. Abnormalities in metabolic processes, as the avitaminosis and xanthomatoses.
6. The benign or malignant neoplasias.

Thus, we have seen that the oral mucous membranes may exhibit, from an objective point of view, most of the primary lesions as they occur on the skin, with a few secondary modifications. And, just as on the skin, there may be erythema, edema, maceration, erosions, scars and the like.

The subjective symptoms of disease in the mouth are, for the most part, soreness, tenderness, burning, stinging, pain, bad taste, and paresthesias. The treatment of oral mucous membrane lesions depends on the basic tenet of good medical practice: do not attempt to treat a symptom as such, but attempt to see that the underlying disease is properly managed.

URTICARIA

With this general background, let us take up some specific conditions. First let us consider urticaria. In this condition we must always keep in mind the peculiar fact that two individuals will not react the same to an allergen; and that even in one individual there will be great variations in the way his skin responds in different areas of his own body. Often, a person who shows marked cutaneous response to a specific allergen may show no effect whatever in the mouth, and of course, the reverse is true. Thus, skin testing for a mucous membrane response is not always reliable. In this connection, we should note that the hormonal, nutritional, and hematological background of an individual modifies his response to most diseases. So also do stress factors, which actually come under the heading of hormonal influences. But stress influences the type and degree of most disease manifestations, and the degree of influence of such stress is in turn dependent upon a great number of factors.[1] We may list among such factors those of heredity, predisposition, state of nutrition, ovulation cycle in women, hygiene, fatigue, chilling, nervousness; emotions such as fear, anger, and hostility; fever, trauma, virulence of pathogens, diet, habits, and even the amount of salivation.

Urticaria of the mucous membranes is diagnosed by the formation of wheals, or hives, which are fleeting, non-permanent lesions, and which are changeable in color, distribution and form. They may be erythematous or whitish and frequently have a surrounding halo of hyperemia. Agents which usually produce urticarial lesions or edema of the mucous membranes are the same as those producing such manifestations on the skin. These are drugs, serums, food, infections, parasites, menstrual abnormalities and the like. The treatment consists of ice cold mouth washes and the administration of antihistamines or steroids.

DRUG ERUPTIONS ON ORAL MUCOSA

Drug eruptions as manifested on the oral mucosa fall into two main categories:

First, the transitory, and second, the so-called "fixed" or permanent lesions. They may be of almost any nature, running the entire gamut of primary lesions and many of the secondary ones. They are caused from an intolerance to drugs or chemicals which may have been ingested, inhaled, or injected. Although there is no drug or chemical to which someone somewhere will not be sensitive, the commonest offending drugs include the salicylates, aspirin, antipyrine, acetanilid groups; the gelatins and oils used as vehicles for injections; barbiturates; the halogens, especially bromides and iodides; phenolphthalein, which is commonly used in cathartics and in pink icings; quinine; sulfa drugs; antibiotics; and the opiates as codeine, morphine and demerol. (Figs. 2 and 3.) In addition, many persons show an intolerance for heavy metals so frequently used medically or commercially. All dentists know that poor oral hygiene

Fig. 2. Drug eruption. Diffuse mucosal edema. (Aminopterin)

Fig. 3. Drug eruption. Massive gingivitis due to Dilantin.

predisposes to deposition of these heavy metals in the mouth. Of these metal deposits, bismuth is probably the most common. Since the advent of penicillin in the treatment of syphilis the use of bismuth has been markedly reduced in medicine. But it is still used by some doctors in the treatment of warts and lichen planus. Bismuth deposits show the classic black line along the gingival margins. The other metals are deposited in the same place, except that lead produces a gray, not black line. Copper produces a green or red line, and silver forms a distinctly violet line with a metallic luster. Frequently, the other oral manifstations are somewhat similar, consisting of altered taste, salivation, stomatitis in the form of erythema, vesiculation or of bulla formation and ulcerations. The differential diagnosis depends on actually finding the metal in the excretions or in the skin, and depends too upon the rest of the systemic symptoms and findings. There may be neuritis, dermatitis, gastrointestinal difficulties, nephritis, or mental changes. The dentist should keep in mind the possibility of such poisonings. Lead poisoning may be incurred by lead smelters, or by persons working in the manufacture of storage batteries, insecticides, and brass making; or by those in certain printing and painting processes. Domestically, lead water pipes and lead paint are the commonest sources of lead poisoning.

Mercury poisoning produces severe oral symptoms, with a diffuse stomatitis, marked salivation, edema and ulceration in the mouth. There are frequently cutaneous eruptions which show any one or all of the primary types of skin lesions at the same time. When this happens the condition is called *erythema multiforme,* no matter what the cause of the eruption may be.

Arsenic rarely produces oral lesions. The only oral complaint may be an excessively dry mouth. The skin, however, will show any of several manifestations, from multiple horny growths, to multiple slow growing cutaneous malignancies, to a complete exfoliative dermatitis.

Persons using chromic acid sprays occupationally may develop chrome poisoning. This produces ulcers of the nasal septum and of the mouth. The mucosa is inflamed and oozes a thin watery discharge.

Gold is an important source of systemic poisoning. It is used medically as gold salts for certain chronic skin diseases, notably lupus erythematosus. As with bismuth, gold is being supplanted by newer drugs. The cutaneous manifestations may be of any type from localized patches of erythema to any of the other primary forms of lesions, or even to a generalized exfoliative dermatitis. The oral mucosa may also show a variety of types of eruptions varying from dryness and cracking of the lips to vesiculation and ulceration; or there may be just a faint purplish pigmentation of all the mucous membrane.

The so-called "fixed" drug eruptions may occur in the mucous membranes of the mouth just as they do on the skin. Most fixed drug eruptions consist of the formation of a placque which is a sharply defined, slightly elevated

lesion of almost any size. The lesion persists for a few days and gradually goes away, leaving a pigmented area, only to go through the same active process in the same location when the drug is repeated. Other fixed eruptions may be vesicles or bullae recurring in the selected area.

ERYTHEMA MULTIFORME

An entirely unrelated group of systemic diseases which manifest themselves in the oral mucous membranes are some of those which are blood-borne. One of the most important of these is the classic *erythema multiforme*. This is characterized by a bullous eruption of one or of all the mucous membranes. It is an acute inflammatory disease and the oral mucosa shows purplish red macules, papules, nodules, vesciles or bullae. The lesions may be grouped or discrete. A similar condition may occur, as mentioned earlier, as a drug eruption. But a specific form of it occurs, and is called the erythema multiforme of the Hebra type. This affliction recurs a number of times with great regularity either in the spring and/or fall. It is accompanied by similar lesions, perhaps over the entire body, but usually on the palms and flexor surface of the forearms. Here the primary lesion is a vesicle or bulla which enlarges, then involutes, only to have a new vesicle appear in the center in a superimposed fashion, giving a peculiar appearance of concentric rings or 'iris" lesions. The etiology is supposedly a combination of virus and allergy. The treatment is steroids. When this disease is unusually severe and generalized, accompanied by fever and prostration, is it called Stevens-Johnson syndrome.

BEHCET'S DISEASE

Another recurrent bullous eruption of the mouth is the condition called the "Triple Symptom Complex" or, Behcet's disease. In this disease there are papulovesicular patches on the buccal mucosa, the palate, the tongue, and sometimes also on the conjunctivae, and usually also on the genital mucosa. The placques quickly become denuded and reveal an oozing inflammatory base, often covered by a pseudo-membrane. The condition is notoriously recurrent for a number of years despite easy response at the time to antibiotics and to steroids. The etiology is not known.

DERMATITIS HERPETIFORMIS

The second of the "big three" of the bullous dermatoses aside from erythema multiforme, is dermatitis herpetiformis. Occasionally, this affliction presents bullous mucous membrane lesions indistinguishable from those of erythema multiforme. The diagnosis is made from the composite picture with the cutaneous lesions and also from the response to therapy. The skin lesions are characteristically grouped, which is the main diagnostic feature of all herpes lesions. These groups are widespread and show deep excoriations as evidence of their intense pruritus, or itching. When the lesions heal there are small scars to mark the site and often there is post-inflammatory pigmentation. Scars and pigmentation are obvious signs of chronicity. This long duration is one of the distinguishing features of dermatitis herpetiformis. The condition lasts from five to twenty years, the average being fifteen years. The cause is supposedly bacterial or viral plus the usual suggestions of nervous factors. In short, we do not know the cause of this not uncommon condition. It does not respond to the steroids or to antihistaminics. To date, the only effective means of control is daily administration of small doses of sulfapyridine over a period of years.

PEMPHIGUS

The third and worst of the big three bullous diseases is pemphigus. This condition almost always attacks all the mucous membranes. In cases where the mucous membranes are not involved the dermatologist usually feels more cheerful regarding the effectiveness of therapy. This condition is often seen first by the dentist. It occurs in either an acute fulminating form difficult to control and often terminating fatally, or it may occur in a less violent, or chronic form. We name pemphigus according to how it looks on the skin. If it is "ordinary," or the usual recurrent large bullae which come and go continuously or in cycles, we call it the common

or vulgar form: pemphigus vulgaris. If the skin is involved so continuously that almost all the top layers of epidermis keep lifting off, or exfoliating, we call it pemphigus foliaceus. If the lesions tend to dry and the debris continues to pile up we call it pemphigus vegetans. Strangely, although the entire skin may become involved with pemphigus, the skin does not tend to scar. The main differentiating sign between pemphigus and the other bullous diseases is the presence in pemphigus of the "Nikolsky" sign. Here, one may place the finger firmly on normal appearing skin immediately adjacent to a bullous lesion and by moving the finger solidly the skin can be felt to slip off the underlying tissues. As mentioned before, the eruption frequently begins on the oral mucous membranes and may be there for months before cutaneous lesions appear. The primary lesion is usually a flaccid fragile bulla occurring on the gums, and when it is ruptured the resulting erosion is unusually slow to heal. Eventually there is extensive denudation and ulceration. The patient is sick when it progresses this far and has difficulty swallowing. The debris often dries because he keeps his mouth open. There is almost always a characteristic peculiar fetid odor to the pemphigus patient. The etiology is not known, but the disease acts like a severe toxemia probably of bacterial or viral origin, influenced by the individual's hormonal status. The disease occurs chiefly in men or women late in middle life or in late life. Occasional cases of it are seen in young persons, but these are almost 100% in young women either pregnant or just over a pregnancy, often resulting in the death of the mother. It is sometimes transmitted to the infant, who proceeds slowly to get well. This disease is controlled quite well in this age of steroids, but an occasional patient cannot be saved by any means at our disposal today.

LUPUS ERYTHEMATOSUS

Lupus erythematosus is one of the collagen group of diseases, that is, related to the rheumatoid conditions; and it seems to be definitely on the increase. This disease occurs in two main forms also: first, the "discoid" or localized

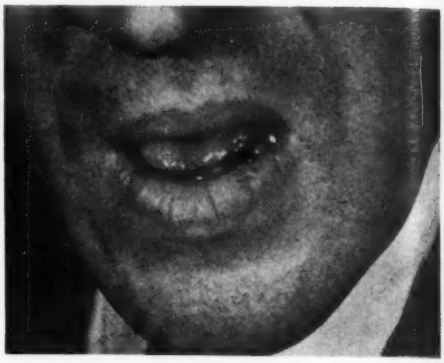

Fig. 4. Discoid Lupus Erythematosus of lower lip.

type; (Fig. 4.) and second, the systemic or disseminate type. Here again, the dentist may be the one to see the original lesions over a period of months or years before cutaneous or systemic symptoms appear. Usually the discoid form remains just that and rarely disseminates. The mucosal lesions appear on the lips, buccal surface, the tongue and on the hard palate. The lesions may appear only as ill defined erythematous macules, which are either discrete or confluent. There is usually a silvery or whitish adherent membrane, removal of which causes bleeding. At the margins are fine white radial lines and dilated blood vessels. There may or may not be an associated inflammatory areola. On the buccal surface there is a tendency toward central depression as seen in partial scar formation. White irregular scars eventually appear. On the tongue, the lesions are usually smooth reddened areas; or, they may be patches of white roughened epithelium. The diagnosis of the chronic form is made chiefly by finding the characteristic skin lesions. These are almost always located about the face, ears and scalp. They are sharply defined "discoid" areas, usually about dime sized, sharply outlined, with slight evidence of retraction. The pores are characteristically widened and the scale dips down into each of the little openings, so that if the scale is lifted up it is seen to have a classic "carpet tack" appearance on the under surface. In the colored person the lesions are usually dark brown to shiny black. In the white, the color

varies. The lesions heal by scarring as a rule. The classic distribution is on the scalp, in the ears, and usually, not always, on the upper portions of the face, especially over the malar eminences and on the temples. In the disseminated variety, the skin lesions are not histologically specific as in the discoid type. But there is more frequently than not a flush over the malar eminences and bridge of the nose in a continuous pattern forming the so-called "butterfly" erythema. In many cases of disseminated lupus erythematosus, the oral mucous membranes are covered with bullae. The diagnosis is made by the finding of the specific lupus erythematosus in the blood or bone marrow; by classic biopsy findings in the discoid form; by a reversed albumin-globulin ratio in the disseminate form, along with fever, high sedimentation rate and leukopenia. Here again, the cause of this disease has not been determined. The disseminated form, we used to learn, occurred chiefly in girls from fifteen to twenty-two years of age. Now we see it also in people in middle age, men as well as women, colored or white. The preponderance is still chiefly in women, however. The discoid form occurs in men almost as frequently as in women. The disease is controlled according to the type or stage of it by steroids chiefly, and by a wide variety of quinine derivatives, notably aralen, chloraquin, and atabrine.

PERIARTERITIS NODOSA

Periarteritis nodosa is another of the collagen group of diseases. The mucosa shows any or all of a wide variety of lesions from simple erythematous macules to purpuric macules, to bullae or ulcers. The patient shows general findings of cutaneous lesions from purpura to nodules, to bullae or urticaria, with subcutaneous, painful nodules along the course of a superficial artery. There is marked vascular damage and the patient shows moderate general malaise; remissions and exacerbations of fever; tachycardia, rheumatic pains, weakness, hypertension and albuminuria. The treatment consists chiefly in the use of steroids, salicylates and hypotensive drugs.

Aside from this group of diseases there is a vast number of metabolic disturbances which cause lesions of the oral mucous membranes. Among these are the endocrinologic conditions you heard about earlier today, including acromegaly, myxedema, the extensive variations in cholesterol and the xanthomatoses. Some of these metabolic conditions are congenital. One is Gaucher's disease, a familial disease occurring chiefly in Jews. The oral mucosa displays gray to brown streaks and patches, which are also seen in the conjunctivae and on the genital mucosa. There is papillomatous hypertrophy of the gums and easy hemorrhage. The skin has a bronze color and there is enlargement of the spleen and liver. Bone lesions and leg ulcers may occur. The disease is usually fatal in children and may involute spontaneously in adults. Nieman-Pick disease is also a Jewish familial disease. All the mucous membranes are involved, and especially those of the mouth, which develop a yellowish tan color. The exposed surfaces of the skin show a peculiar yellow color and may be pale, wrinkled and exhibit profuse sweating. The subcutaneous fat disappears. There is liver and spleen enlargement. Hand-Schiller-Christian disease is usually seen in children. There is gingivitis and stomatitis, chiefly in the form of recurrent aphthae of the gums and buccal mucosa. The diagnosis is based on the classic findings of diabetes insipidus, cutaneous xanthomas, and exophthalmos. Letterer-Siwe disease is believed by many to be a form of Hand-Schiller-Christian disease. It is fatal but not supposed to be hereditary. The condition is rare. The oral mucous membranes show coated ulcerations with raised edges, and there are scattered white papules over the palate, which has an overall yellowish gray coating. The patient shows systemic signs as well, such as hemorrhagic diathesis, purpura, possibly porphyria, and perhaps liver and spleen enlargement. There may be tumor formations in bone, liver, skin and bone marrow. The prognosis is fatal.

Systematized amyloidosis results in mucosal manifestations. There is a disturbed protein metabolism. The cause is unknown. The skin shows either discrete or confluent papules of a shiny, smooth, waxy character. In the mouth there is glossitis and macroglossia. Later, furrows develop in the tongue and some small trans-

luscent macules appear. These are followed by patches having a purulent to necrotic appearance. There is usually a great deal of pain in the tongue. Treatment to date is completely unsatisfactory.

Pregnancy is often accompanied by gingivitis, as is dysmenorrhea. The gums become red and bleed easily. The gingival margins become swollen and develop bulging folds. The condition is believed to be associated either with some obscure hormonal effect or with a combined vitamin and calcium deficiency.

Porphyria is a congenital metabolic difficulty. In this condition there are often vesicular and bullous lesions on the gums and lips with erythrodontia. If this disease appears in its acute form there is an associated cycle of intermittent episodes of serious gastro-intestinal symptoms. There are also nervous and/or mental symptoms. The congenital type of porphyria begins in infancy and exhibits the erythrodontia and associated cutaneous light sensitivity. The chronic form of porphyria occurs in adults in whom there is serious liver damage, such as occurs in alcoholism, or following the use of other hepatotoxic drugs and agents.

Sjogren's syndrome, known also as Gougerot's syndrome, or xerodermatosis, is believed to be related to vitamin-mineral-hormonal deficiencies. It occurs in post-menopausal women and involves all the mucous membranes, but especially the eyes and mouth. There is a dry type of keratoconjunctivitis, a dryness of all the membranes of the nose and throat with a red, dry tongue. There are deep longitudinal furrows, inadequate saliva, perleche, commissural fissures, often fluctuant salivary and parotid glands, glossodynia, and impairment of taste. The skin is excessively dry, the nails, hair and teeth all become fragile and broken. Treatment is aimed at all the involutional factors.

Certain hypertrophic manifestations of the oral mucous membranes are actually systemic in origin. Among these is a condition called acanthosis nigricans, either the juvenile or adult form. The juvenile form is believed to be an endocrine (pituitary-adrenal) problem, whereas the adult form is almost always a peculiar form of toxic dermatitis indicating an internal malignancy, usually an adenocarcinoma. Hyperpigmented, yellow-brown to black velvety papules appear in the folds of the skin at the neck, groin, elbows and elsewhere. In the mouth, the lesions consist of thickening of the buccal mucosa and multiple warty or condylomatous growths on the tongue, lips and epiglottis, resembling condyloma accuminata.

Among the pigmentary disorders, omitting Addison's disease, we may mention several which show lesions in the mouth or on the lips. Vitiligo is a disease in which a variable number of depigmenting areas appear on the face, extremities, or body. The cause of this condition is still not known and it frequently makes the first appearance on the vermillion border of the lips. The treatment today consists of the use externally and internally of 8-methoxypsoralen in addition to frequent ultraviolet exposures. Unfortunately, the results to date have fallen below our hopeful expectation, but some persons have been definitely helped. Kaposi's sarcoma is another disease, the cause of which is not known, and which displays freckle-like hyperpigmented macules over the buccal surfaces. The cutaneous lesions usually appear first on the legs or on the arms, and consist of reddish to purple tumors and nodules. Internal, and especially cardiac lesions, eventually occur.

Jegher's syndrome is a peculiar hereditary condition, occuring in both sexes, and characterized by generalized intestinal polyposis with multiple melanin macules involving the gums, hard palate, buccal mucosa and lips. Similar macules, darker than freckles, may occur on the hands, dorsa of the fingers and on the toes and feet. No treatment is advised for the mucous membrane lesions.

SYSTEMIC INFLAMMATORY DISEASES

Among the systemic inflammatory diseases causing oral mucous membrane lesions are sarcoid, psoriasis, scleroderma, lichen planus, trichinosis, recurrent aphthae, pityriasis rosea, lichen nitidus, probably transitory benign placques of the tongue, and periadenitis mucosa necrotica recurrens. (Fig. 5.) The latter condition occurs on the buccal mucosa, the larynx, and vulva. It begins as small, smooth, red, hard, nodules

Fig. 5. Tip of Tongue. Periadenitis mucosa necrotica recurrens.

which undergo central necrosis and sloughing. A plug separates out and a crater-like depression remains and scars over. The condition is associated with extreme pain and is self limited to six or seven days. It responds to the broad spectrum antibiotics. The cause of the transitory benign placques is unknown. They occur singly or in multiples over the dorsum and sides of the tongue anterior to the circumvallate papillae. The patch spreads out and becomes angular. The center is smooth and redder than normal mucosa. The border of the lesion is slightly elevated and is sharply defined, with no surrounding reaction apparent. The ring formations constantly change their configuration and are transient, only to recur. Treatment consists primarily of improving oral hygiene, improving the nutritional status of the patient and using antibiotics and steroids.

In trichinosis, the tongue and especially the muscles of mastication are involved. The soft palate is edematous and shows petechial hemorrhages. There are usually signs of systemic infestation by the time the mouth shows such extensive changes.

Pityriasis rosea is probably due to a virus. This is a rather common skin eruption which comes on like chicken pox, except there is very little malaise. On rare occasions it shows mucous membrane involvement. The lesions tend to be flat, faintly scaling papules on the skin. These are characteristic, in that they assume an ovoid shape and follow the lines of cleavage over the torso. The eruption classically involves the trunk, the upper arms and neck only; occasionally, lesions occur on the dorsal surface of the tongue and are slightly papular. The papillae may also be swollen. The diagnosis is made on the basis of the cutaneous eruption.

Lichen nitidus is an interesting condition. It occurs primarily in children and consists of clusters of tiny papules usually skin-colored to whitish. They may be scattered anywhere on the trunk and extremities, usually on the extensor surfaces. The tongue and buccal mucosa may also show these small rounded papules. There is supposed to be some relation between this condition and a type of immunity reaction to tuberculosis. This has not been proved. However, treatment is not immediately effective. With strict attention to nutrition, the child usually exhibits no lesions in about one to two years.

Mucosal lesions may be seen in any of the lymphoblastomas. The lesions are usually of the non-specific type, presenting any of the primary types of lesions which can develop in a general toxemia. Occasionally, specific infiltrations do occur. The monocytic type of leukemia seems to show mucosal lesions more often than do the other forms of this group of diseases. Even mycosis fungoides has been reported to appear on the tongue.

Time is short and I will not go into detail regarding the many systemic fungus diseases with oral lesions. Suffice it to list among them:

Actinomycosis, Blastomycosis, Coccidiomycosis, Geotrichosis, Histoplasmosis, Moniliasis, South American Blastomycosis or Paracoccidioidal granuloma, Sporotrichosis, European Blastomycosis, also called Torulosis or Cryptococcosis. Briefly, most of these infections produce granulomatous lesions, often mucocutaneous, usually nodulo-ulcerative. The diagnosis is dependent upon microscopic or cultural identification.

Another group of systemic, yet specific infections includes diphtheria, leprosy, tuberculosis, the latter either as tuberculosis cutis orificialis, primary inoculation tuberculosis, or lupus vulgaris and tuberculosis verrucosa cutis. And, among these systemic specific infections are certain manifestations of gonorrhea, tularemia, and glanders.

Diphtheria lesions occur on the lips, buccal mucosa, commissures and pharynx. The lesions

assume many forms but the most characteristic is the multiple ulcerations with a tough leathery adherent membrane. Sometimes the dentist might recognize the early signs in the prodromal stages.

There are various forms of leprosy, but the one which attacks oral mucous membranes is the lepromatus type. Here, the lesions are red patches with raised edges, nodules, ulcers and diffuse infiltrations involving first the hard palate, then the tongue, the posterior pharyngeal wall and the vulva. The nasal mucosa is also usually involved. Diagnosis is based on recognition of the oral findings, on the cutaneous lesions and on laboratory confirmation. In leprosy, there is usually a positive serology, inverse albumin-globulin ratio, increased sedimentation rate, and bacilli in nasal smears and in tissue sections.

In tuberculosis cutis orificialis, the tongue is most commonly involved, but the lips, commissures, gums, cheeks, and palate are occasional sites. The disease is actually a contamination of the mouth from pulmonary tuberculosis. The lesion is classically a rapidly developed shallow ulcer. This is painful and has a punched-out appearance with a slightly irregular margin; but there is no undermining of the edge of the ulcer. The base of the lesion is granulomatous and is covered with a thin watery discharge. Diagnosis is made by smears or by the histopathology of the lesion; or, sometimes, by recovery of the organism after animal inoculation.

Primary inoculation tuberculosis occurs as a rule following the kissing of a person with tuberculosis. The lesion looks like a syphilitic chancre and is a hard indolent ulcer on the lips or tongue, with non-tender regional lymphadenopathy. Biopsy and cultures prove the diagnosis. Lupus vulgaris and tuberculosis verrucosa cutis develop, as a rule, on the skin and involve the oral mucous membranes, especially the lips and buccal mucosa, by extension.

Gonorrhea may occur as a stomatitis in the new-born resulting from infection acquired during birth. There is a rare form of systemic gonorrhea which erupts into a dermatitis called keratosis blennorrhagica, in which there are peculiar horny, cone-shaped lesions all over the skin. In this condition, mucosal lesions also appear, especially on the buccal, palatal, and lingual surfaces. Here, no horny formation can exist, so the lesions look superficially denuded and oozing, although they are discrete and multiple. They may or may not have a pseudomembrane over them. The diagnosis is made by recognizing the classic dermatologic lesions and by bacterial studies.

Tularemia of the mucous membranes begins with a chancre-like lesion at the site of inoculation also. Differentiation from other such lesions is done by special agglutination tests or by recovery of the organism following guinea pig inoculation. Streptomycin is the treatment of choice.

Glanders is a systemic infection acquired from horses. It is a severe disease, either acute or chronic. The nasal mucosa shows the most characteristic eruption, presenting a profuse catarrhal or purulent discharge. But the ulcerative lesions often occur on the lips and oral mucous membranes, frequently preceded by pustules. The diagnosis is made by the cutaneous pustular and ulcerative lesions, typhoid-like general symptoms and by bacteriologic tests.

Various protozoan diseases affect the oral mucous membranes. One is Leishmania, of the Brasiliensis type; another is the Donovan type, usually called Kala Azar. In the Brasiliensis, between 10 and 20% of all the cases show mucous membrane lesions. These are severe and persistent ulcerations of the palate, larynx and nose, in addition to those on the lips and buccal surfaces. Kala Azar produces an actual gangrene of the mouth and a thickening of the lips.

Yaws, or Frambesia, resembles syphilis almost identically except that in the early stages yaws avoids the mucous membranes. A very small percentage of cases will show secondary lesions resembling the secondary stage of syphilis. But the tertiary lesion of yaws is a severe mutilating ulceration beginning on the hard and soft palates, often proceeding to destroy or to scar the entire face. This condition is mainly a tropical disease and in its tertiary phase is called "gangosa." It responds to the same treatment as does syphilis.

Virus diseases also affect the oral mucous

membrane. One is the foot and mouth disease which is a self-limited, acute condition with fever, chills, headache, muscular pains and malaise. The lips become painful, hot, bright red and dry. The area later becomes covered with vesicles which proceed to rupture, leaving a raw, painful surface, which becomes covered with a grayish exudate. Sometimes the hands show similar lesions The feet may be involved but this is rare. The diagnosis is made usually by the type and distribution of the lesions, the sequence of events, and a knowledge of the presence of the disease in the community. The disease often is not diagnosed at all and such a diagnosis depends largely on thinking of it.

Herpes simplex, which usually is localized, may involve multiple areas either on the mucous membranes or even the skin. There is a generalized condition which is generalized herpes simplex, called Kaposi's varicelliform eruption. The skin is widely involved and herpetic lesions are on the oral mucosa.

Herpes zoster occurs uncommonly in the oral mucous membranes. One of the classic diagnostic features here is the presence of severe pain or neuralgia even before any lesions appear. The lesions are vesicular and grouped as in all herpetiform eruptions and characteristically form in groups along the distribution of a nerve.

Lichen planus, supposedly a viral disease and perhaps related somehow to stress factors, displays a wide variety of whitish papules and placques anywhere on the oral mucous membranes. Diagnosis is made on the basis of the cutaneous lesions which are characteristically on the flexor surfaces of the forearms, although they may be generalized. They are papules, discrete and definitely violet colored, glossy and angulated. Treatment for years has been bismuth, but no treatment is really adequate to date. The condition is self-limited, from a few months to many years. (Fig. VI)

Venereal diseases notoriously affect the mucous membranes. Syphilis in its primary stage forms a chancre which not infrequently occurs on the lips or tongue. The secondary stage

Fig. 6. Gingival Lichen Planus.

shows the classic mucous patches, which are macerated mucosal forms of the generalized cutaneous papulo-squamous eruption. The tertiary lesion is the gumma, which, while not infectious, is a destructive granuloma.

Malignancies occurring in the oral mucous membranes may represent systemic involvement. Metastatic carcinoma, sarcoma, and melanoblastoma are among these.

CONCLUSION

In conclusion, we may say that almost any systemic condition which shows a cutaneous eruption may show oral mucous membrane involvement. This covers the entire field of allergic responses, blood-borne bacterial and viral diseases, metabolic, endocrine, deficiency, and neoplastic diseases. It includes congenital diseases and pigmentary disorders as well as inflammations, nervous disturbances, fungous and protozoal diseases, and venereal diseases. A great many of these may have their first and occasionally their only manifestation on the oral mucous membranes. By the knowledge of the similarities and differences in reaction between the oral mucous membranes and the skin, the dentist may be able to classify many of these diseases. He should be aware of his tremendous responsibility to the patient and to the community in the early detection of disease and should direct his careful attention toward mucous membrane as well as dental abnormalities.

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MORBOS SYSTEMIC E LOR MANIFESTATIONES IN LE MEMBRANAS MUCOSE DEL BUCCA

Dr. med. Alice E. Palmer (M.S.)

SUMMARIO IN INTERLINGUA

Il existe un grande numero de morbos systemic in que le oral membrans mucose es implicate. Illos include allergias, disordines del metabolismo, morbos de carentia, e congenite, pigmentari, neurologic, bacterial, fungal, protozoal, veneree, e systemico-neoplastic morbos.

Quasi omne condition systemic que monstra un eruption cutanee pote monstrar un implication de oral membranas mucose. Iste criterio imbracia le integre campo de responsas allergic, de morbos bacterial e virusal a dissemination sanguinee, e morbos metabolic, endocrin, carential, e neoplastic. Illo imbracia morbos congenite e disordines pigmentari tanto ben como inflammationes, disturbationes nervose, morbos fungal e protozoal, e morbos veneree. Un grande numero de iste morbos pote haber lor prime (e a vices mesmo lor sol) manifestationes in le oral membranas mucose. Per cognoscer le similaritates e le differentias inter le oral membranas mucose e le pelle, le dentista pote succeder a classificar multes de iste morbos. Ille debe esser conscie de su tremende responsabilitate verso le patiente e le communitate quanto al precoce detection de tal morbos e debe prestar le plus meticulose attention a anormalitates del membranas mucose tanto ben como a anormalitates dental.

ORAL MANIFESTATIONS OF ENDOCRINE AND NUTRITIONAL DEFICIENCIES*

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The subject chosen for this discussion is a large one which offers some difficulties of division into oral and dental manifestations. I will confine my remarks to clinical findings in man. I shall not attempt to interpolate experimental findings from laboratory animals into those findings of man. The findings of animals under unusually severe conditions cannot be used to indicate that the same findings exist in humans under much less severe conditions. (Even assuming the metabolism, reactions, etc., are the same for the different species). This interpretation of findings in laboratory animals as typical of man has resulted in much confusion in both medical and dental literature. An effort will be made to avoid adding to that confusion. I will discuss first the changes associated with endocrine disturbances and then the nutritional changes. The first endocrine gland to be discussed will be the pituitary gland.

This is a complex gland producing numerous factors which effect various parts of the body, especially other endocrine glands. The anterior lobe produces growth hormone, gonadotrophic, thyrotropic, adrenocorticotropic (ACTH), lactogenic, diabeticogenic hormones and perhaps other factors. The posterior lobe produces two hormonal substances: Pitressin, which increases blood pressure, and Pitocin, which stimulates contractions of smooth muscle. Further details regarding the function of the pituitary gland can be found in any text on endocrinology.

In the discussion of any endocrine gland it is always necessary to consider two states of activity: Underfunctional state (hypofunction), and overfunctional state (hyperfunction). It is also of importance to consider the age at which the altered functional activity occurred.

Both the hypo and hyperfunctional states of the pituitary will be considered, prepubertal and postpubertal.

THE PITUITARY GLAND (GROWTH HORMONE)

Hypofunction before puberty: Prepubertal hypopituitarism produces dwarfism of the Lorain type. There is symmetrically diminished development of all parts; the body proportions are normal. Impairment of development involves the teeth as well as the skeleton. Impaired development of carpal and metacarpal bones and of the teeth are correlated so the individual may have a skeletal and dental age less than the chronological age. This is not only manifest in formation, but also in eruption so that teeth erupt late. Although some authors have described gingivitis associated with dwarfism, it is not of higher incidence or greater severity in dwarfs than in normal individuals. Due to the lack of facial development, there may be some crowding of teeth.

Hypofunction after puberty: This usually occurs in females during the reproduction period and is characterized by marked loss of weight and atrophy of all parts. This is called Simmonds disease.

The oral changes are those of atrophy and lack of resistance of the periodontal membrane to functional load; lack of resistance to periodontal disease.

Another evidence of hypofunction is seen in Fröhlich's syndrome initiated early in life by tumors or surgery in the pituitary area. The physical changes are characteristic, but the dental changes are secondary. The patients frequently have diabetes insipidus and take a part of their fluids in soft drinks; therefore, they have a high caries rate. No other dental defects have been observed.

Hyperpituitary function (Prepubertal): Hyperfunction during this period leads to giantism and precocious development. The skeletal age and the dental age may be above the chronological age; thus the individual may show early

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eruption of teeth. There is usually spacing of the teeth due to the excessive size of the jaws. Of significance to dentistry is the fact that these individuals have little resistance to infection, and this should be considered when dealing with dental infection.

Hyperpituitary function (Postpubertal): Hyperfunction during this period leads to acromegaly. The continued growth in this condition takes place in the peripheral portions of the skeleton so the hands and feet show marked enlargement, as do the skull and jaws. The most peripheral portions of the skeleton, of which the mandible is a part, show the greatest growth. This results in spacing of the teeth and the change in occlusal pattern with the development of prognathism. The tongue is enlarged, and may by pressure, contribute to the spacing of the teeth. The lips are also heavy. These acromegalic changes may be superimposed on giantism if the onset takes place during early adolescence.

THE THYROID GLAND

Prepubertal hypothyroidism results in cretinism which may be congenital or acquired. However, both produce similar changes, so they will be discussed together. There is delay in growth so the dental and skeletal age are lower than the chronological age. The dental age is usually less than the skeletal age. The effects on the teeth may vary according to age on onset. They are more severe in the cretin. Some investigators have indicated that there is a high caries activity, but this depends on the diet and the presence of poor oral hygiene. High caries activity is not a constant finding. Gingival changes are usually present. The tongue is oversized and the lips thick. Much has been said about high caries activity in thyroid disease, but this is usually due to an anticipated observed relationship. Because there are alterations in metabolism, it is believed that the teeth are poorly formed and thus susceptible to caries. Observations of hypoplastic teeth indicate that this is not true. Individuals with hypothyroidism may be on a diet high in carbohydrates. Because of decreased salivary flow, and thus decreased washing action of the saliva, carbohydrates may accumulate in the mouth. Hypothetically, because of the relationship of caries and carbohydrates, decreased salivary flow might contribute to a high caries incidence in individuals with hypothyroidism. However, the incidence of caries is no higher in these individuals than that which may be seen in children who do not have hypothyroidism. The caries incidence may also appear higher because of retention of deciduous teeth. It should be remembered that most cases of severe hypothyroidism are treated by physicians who, because of their limited dental observations, do not realize the ravages of caries in normal individuals; and therefore, when they see a high caries rate in children with hypothyroidism, they attribute it as a symptom of thyroid disease.

Hyperthyroid function: The most important finding in these individuals is nervousness, emotional instability and tachycardia, all of which goes to make them poor dental patients. Also the patient with extreme thyrotoxicosis is one which should not have surgical procedures performed because of the possibility of precipitating a thyroid crisis. The administration of local anesthesia containing adrenalin may exaggerate the tachycardia and arrhythmia which these patients exhibit. This results in apprehension and syncope. These individuals respond poorly to periodontal therapy. Both hyper and hypothyroidism are attributed as factors in periodontal disease. Although they are not initiating factors, they may alter the course of pre-existing periodontal disease because they alter the metabolism, and thus the ability to repair.

THE PARATHYROID GLAND

Hyperparathyroidism: There is an increased mobilization of calcium and phosphorus from bones resulting in osteitis fibrosa cystica, loose teeth, alveolar bone loss (not from crest), and giant cell tumors, both central and peripheral.

The textbooks indicate that the jaws are a special site of predilection for demineralization which results in increased mobility of teeth due to destruction of alveolar bone. The loss of lamina dura is pointed out as a characteristic x-ray finding. This is an error which has been perpetuated extensively in the literature because of the failure to distinguish between the bone loss from periodontal disease and that due to

hyperparathyroidism.

The patients are frequently in the age group showing periodontitis with loss of bone from the alveolar crest. It is this loss of bone which is incorrectly attributed to hyperparathyroidism. The bone loss in hyperparathyroidism involves medullary as well as alveolar bone. It has no special predeliction for alveolar bone. The areas of bone loss are focal demineralization which involve both alveolar process and alveolar bone. This occurs in the substance of the jaw, and therefore involves alveolar bone in areas other than the crest. The loss of a large amount of alveolar bone in the lateral and apical areas results in mobility and drifting of the teeth with an absence of pocket formation. The hyperparathyroidism may occur in a patient having periodontitis. The concomitant finding of the two conditions do not indicate a relationship. However, after a tooth becomes excessively mobile from bone loss due to hyperparathyroidism, there may be as the result of local and traumatic factors, the development of a localized periodontitis. It is in this instance limited to a single tooth or a few teeth in a localized area.

The development of giant cell tumors of the jaws, central and peripheral, are seen in manifestations of hyperparathyroidism. All giant cell tumors, especially those that demonstrate recurrence, should suggest the possibility of hyperparathyroidism and indicate the need for clinical laboratory studies.

Hypoparathyroidism: This condition when present in infancy may be responsible for the production of hypoplastic enamel. Hypoparathyroidism occurring in adult life as the result of renal disease or as the result of the removal of the parathyroid glands during thyroidectomy is of very little dental significance. Comroe states in concluding his discussion of parathyroid glands: "The parathyroid glands evidently play a role in the calcification of the enamel and dentine during the formative period of dentition. Decreased parathyroid activity in childhood may be in part responsible for the hypoplasia of the enamel seen following infantile tetany. Evidence is still lacking to connect dental caries or periodontal disease to parathyroid activity."

THE THYMUS GLAND

This may be of dental significance only when surgery is to be done under general anesthesia in infancy. In this case the individual with a hyperplastic thymus as a manifestation of a thymicolymphatic constitution may die of a thymic death. The presence of thymicolymphatic constitution should always be considered in infancy, and the individual should always be examined by x-ray for evidence of an enlarged thymus. If present, the individual should be treated by irradiation.

THE PANCREAS

Lack of function of the Islets of Langerhans results in diabetes mellitus. The association of diabetes with oral disease has long been recognized, and there has been much written on the subject. The most important dental consideration in diabetes is the association with periodontal disease. The literature leads to the conclusion that diabetes produces specific types of gingival and periodontal disease. It has, however, been shown by Glickman and others that diabetes per se is not the cause of periodontal disease, but that diabetes alters the course of already existing gingival or periodontal disease. The characteristic gingival picture in diabetes is that of swollen, boggy gingiva having a bluish color and a soft consistency. There may or may not be pus in the periodontal pockets which are frequently deep and numerous. The radiographic picture is usually one of excessive loss of bone, and there may be resultant looseness of the teeth. The oral hygiene is usually poor, and there is abundant materia alba and often calculus. These patients frequently develop periodontal abscesses due to deep pockets, heavy calculus deposits, and tooth mobilty. The patient with diabetes should be treated first by an internist to obtain control of the diabetes. Periodontal therapy can then be carried out successfully. It is imperative if surgical treatment is indicated that the patient be maintained in diabetic control during the period of healing.

Much has been written relative to diabetes and dental caries. The consensus of opinion indicates that it is the diet of the individual

rather than the diabetes which is responsible for the increase in caries activity.

THE ADRENALS

These are very complex glands which produce a variety of hormonal substances. Little is known about the relationship of these substances to dental disease. There is some indication that although there may be no frank evidence of adrenal dysfunction present, hypo or hyper activity of the adrenal glands may alter the physical and physiologic characteristics of the individual, and thus have certain clinical significance. In those females having an excess of facial hair, low-pitched voice, and some alterations in menstrual history there is frequently a gingivitis which is characterized by increase in bulk of gingival tissue and marked tendency to gingival hemorrhage. These findings would suggest excessive production of androgenic hormones with alteration of ovarian function, and thus producing the characteristic hemorrhagic gingivitis. This form of gingival disease does not respond well to routine gingival therapy. Other factors in the adrenal is the hypofunction which results in Addison's disease with a characteristic and quite constant oral pigmentation. The appearance of a brownish to blue-black pigment in the buccal mucosa and the dorsum of the tongue is often an early sign in this disease. The individual also may exhibit extensive periodontal disease which shows a rapid downhill progress due to the lack of resistance of the gingival tissues to local irritation and infection.

THE GONADS

The ovaries produce hormonal substances which are related to the menstrual cycle and pregnancy. Alterations in these substances produce oral manifestations.

Changes are seen during pregnancy which suggest that as a result of an increase in progestrin, there is stimulation to the vascular supply of the gingiva that results in the production of pregnancy gingivitis and in some instances, pregnancy tumors. Although this is called pregnancy gingivitis and there is evidence of hormonal stimulation of the gingiva, it should not be considered as being caused by the pregnancy status. It is the superimposed hormonal effect in a patient who had a pre-existing gingival disease. The gingivitis which was present before pregnancy has an altered clinical picture because of the superimposed hormonal stimulation. The only way we can make a diagnosis of pregnancy gingivitis is to know the physiological state of the patient. The marked improvement or cure produced by local treatment during pregnancy is evidence of this relationship.

Similar changes are seen in children at puberty and are associated with imbalance of gonadal hormones. The gingival changes are similar in both instances.

In postmenopausal women there are oral changes associated with reduced hormonal stimulation. One type of change is characterized by hyperplasia of vestibular glands and increased viscosity of saliva. The patient complains of burning of the lips and tongue which they frequently associate with the placement of a restoration or with the wearing of dentures. These patients are frequently highly nervous and have emotional conflicts which aggravate the condition. Usually no clinical signs are present in the tongue or gingiva, and those in the vestibular mucosa are minimal.

Another postmenopausal change is chronic atrophic gingivitis. This usually involves only the gingiva but in severe cases may involve the buccal mucosa. The change is characterized by atrophy of the mucosa with increased cornification. The gingiva is pale whitish in color. The free margin is thin and usually receded. The gingival sulcus is shallow if the mouth is well kept. Pre-existing gingivitis may alter the picture by the presence of inflammatory changes. The only symptoms are the feeling of dryness and roughness of the gingiva. The patient may also have a chronic atrophic vaginitis. The microscopic changes in the two mucosae are the same. The changes may be mild to severe, and in some cases progressive; in others they remain status quo. The patient should be observed at intervals to make sure neoplastic change does not occur.

Chronic desquamative gingivitis is often considered as a manifestation of reduced gonadal stimulation, especially in those patients having

an irregular menstrual history. The gingival changes are characterized by exacerbations and remissions of desquamation. The desquamation is followed by regeneration and hyperkeratinization of the gingival epithelium during which time the patient is comfortable. After a variable interval desquamation occurs and the gingiva again becomes very sore. The exacerbations and remissions cannot be correlated with the menstrual cycle. Although a few patients give a history of an irregular menstrual cycle, many patients with desquamative gingivitis do not. Similar changes may be produced by lichen planus, pemphigus, allergy, infection; and therefore, they must be considered in differential diagnosis.

Testes: Occasionally the male develops pubertal gingivitis which may be produced by the action of testosterone at the time when the gonads are becoming functional. The complexity of the interrelation between the gonadotrophic hormones of the pituitary and adrenals, and those produced by the gonads make it difficult to pinpoint the specific nature of the reaction. It is, however, quite definitely due to hormonal imbalance as is evidenced by the period of onset and the disappearance of the condition with physiologic adjustment. The changes are the same as seeen in pregnancy gingivitis. Granuloma pyogenicum may develop at this time further stimulating the picture of so-called pregnancy gingivitis.

NUTRITION

The problem of nutrition is a complex and difficult one to discuss. There are great variations of opinion as to the part played by the various nutritional factors, especially the accessory food factors.

In order to discuss nutrition we should have an understanding of what constitutes nutrition. It is the summation of the dietary intake, the absorption, the storage, and the utilization of the food factors by the tissues. A frequent error in the discussion of the subject is made when diet and nutrition are considered to be synonymous terms. Diet is the food intake, and nutrition is the absorption and utilization of these food substances. There may be many causes for nutritional deficiency other than limitation of dietary intake. According to Darby, the most common form of malnutrition in the United States is obesity. Florid deficiency states of dietary origin are encountered infrequently in the United States today. Deficiency states are usually of the conditioned type, or as the result of replacement of food by alcohol. The conditional deficiencies are rather complex medical problems, and it should be emphasized that recognition and treatment require more than a cursory dietary history and the empirical administration of vitamins or dietary advice.

In the consideration of vitamin deficiencies it is well to remember certain principles. The absorption, storage, and excretion of vitamins adhere to two broad patterns. The water soluble vitamins (B-C) are diffusible, and examples of impaired absorption are not common. These factors may be stored in amounts to prevent the development of clinically manifest deficiency states over a period of deprivation of four months to a year. They are excreted in the urine, and hence do not accumulate in toxic quantities from excessive doses. The fat soluble factors (A-D-E-K) are absorbed with lipids, and derangement may result when the absorption of lipid is prevented. The body has a great storage capacity for these factors, and may require a year of deprivation to obtain clinical evidence of deficiency. They are not excreted in the urine, so that prolonged, excessive dosage may produce toxic tissue levels.

It would be impossible in the limited time available today to discuss all of the nutritional diseases adequately. We will therefore be able to indicate only certain oral conditions which are most commonly found associated with nutritional disturbances.

The vitamins are the most frequently discussed nutritional factors which produce oral manifestations, so we will discuss them first and in alphabetical order.

Vitamin A Deficiency

This is a soluble fat vitamin necessary for the normal metabolism of epithelial tissues. The dental significance of avitaminosis A is found during the period of development of the dentition. Deprivation experiments in laboratory

animals results in hypoplastic enamel. This occasionally may be true in humans, as has been shown by Boyle and a case reported by myself. Hyperkeratosis of the buccal mucosa in the adult has been attributed to Vitamin A deficiency, but the lack of response to heavy doses of Vitamin A would indicate that it is not a factor.

Vitamin B Complex

Vitamin B_1 (Thiamin) Deficiency—Beri Beri (Polyneuritis, edema)

There may be some changes in the tongue characterized by slight enlargement due to edema and lack of muscle tone. Fungiform papillae are enlarged and hyperemic.

Vitamin B_2 (Riboflavin) Deficiency—Angular cheilosis

Cheilosis of the commissural angles with the development of perleche-like lesions and desquamative cheilosis of the vermillion portion of the lip resulting in reddening and crusting. The tongue is purplish or magenta in color. The filiform papillae are atrophic producing a clean tongue. The fungiform papillae are enlarged due to edema and produce a furrowed character to the tongue. The tongue may be painful and may be the most troublesome symptom. The angular cheilosis should be differentiated from the cases of moniliasis and loss of vertical dimension which produce similar changes.

Nicotinic Acid (Niacin) Deficiency—Pellagra

Painful oral manifestations are early symptoms. There is usually a painful glossitis with intense burning of the tongue. The clinical appearance of the tongue varies from smooth and pale to swollen, beefy-red with numerous areas of ulceration. In the early stages the tongue is clean, and papillae are present. In the late stages there is atrophy and ulceration. The mucosa is atrophic, red in color, and painful. There are shallow ulcers present in advanced stages of this disease. These changes are seen associated with frank evidence of pellagra, and not as subclinical states.

Folic Acid Deficiency—Sprue (Macrocytic anemia)

After an initial episode of diarrhea, oral lesions appear first as a burning sensation of the tongue, and then the development of numerous, small herpetic-like lesions which become small, superficial erosions. The tongue is swollen and the fungiform paillae are enlarged. Angular cheilosis also frequently is a part of the picture.

Vitamin B_{12} is related to pernicious anemia. In pernicious anemia the individual has a burning tongue and atrophic glossitis. The atrophy and the glossodynia start at the lateral borders and progress medially; in advanced cases the entire tongue is involved. The buccal mucosa also may be atrophic in character. The atrophy gives the impression of an exceptionally clean mouth. In some cases icterus may be present and impart a yellowish tinge to the mucosa.

In any discussion of Vitamin B it should be remembered that deficiencies are rarely single deficiencies, but are usually a combination of several factors, symptoms of which may be superimposed on each other producing a confusing picture. This has resulted in much confusion in the literature in describing the clinical manifestations of the various components of the Vitamin B Complex.

Vitamin C Deficiency—Anti-scorbutic (Scurvy)

The effects of Vitamin C on gingival tissue has been considerably exaggerated in both medical and dental literature. A deficiency of Vitamin C has been given credit for producing various forms of gingival and periodontal disease.

The typical scorbutic gingivitis has been described as one characterized by marked enlargement of the gingival tissue with marked tendency to hemorrhage. Hemorrhage into the gingiva produces marked discoloration. Progressive destruction of the supporting tissues results in loosening and loss of teeth. The investigation of Crandon et al. have verified what many have suggested; that the avitaminosis C is not responsible for the gingival disease, but does intensify the pre-existing gingival disease when lack of Vitamin C is superimposed.

Vitamin D Deficiency—Anti-rachitic (Rickets)

Hypoplastic enamel may be produced by lack of Vitamin D during the period of enamel formation. No changes are produced after enamel formation is complete. Some investigators have tried to show that Vitamin D in children

in the posteruptive period gave some protection against dental caries. This, however, has not been well substantiated.

Vitamin K Deficiency

The failure of absorption of Vitamin K results in hemorrhagic disease due to deficiency of prothrombin. This is usually associated with jaundice which can be observed in the oral mucosa as well as the sclera and skin. The chief dental significance is the tendency for hemorrhage following extractions. When jaundice is present, prothrombin level and bleeding time should always be investigated before any surgery is attempted.

MINERALS

Calcium and phosphorous are important minerals for the development and maintenance of calcified tissue. The lack of these minerals, especially in the presence of Vitamin D deficiency, may result in the development of hypoplastic enamel if it occurs during the formative period. However, most of the clinical and experimental evidence indicates that calcium deficiency per se is of no significance in producing hypoplastic enamel. If the diet is inadequate in calcium salts the deficiency is compensated for by withdrawal from the bones, and normal blood levels maintained. If the deficiency is severe, osteomalacia results but dental defects are not evident. This has been shown by the experiments of Fish and the observations of Marshall-Day.

The same may be said for phosphorus, as the two are closely related. Osteoporosis may be associated with reduced dietary intake of calcium and phosphorus, renal disease, and following the menopause. Therefore, evidence of a delicate bone picture should not be interpreted as due to dietary deficiency. This is not a factor in the development of periodontal disease.

Fluorine when present in water supply in more than two parts per million may produce hypomaturation of enamel. This is called mottled enamel because of the numerous opaque areas distributed through the enamel. The teeth in fluorosis may fracture easily and expose dentin which becomes highly sensitive. This condition is observed in areas where there is a high percentage of fluorine in the water supply. The severity and frequency is proportionate to the fluorine concentration.

Iodine deficiency may be responsible for the production of hyperthyroidism which we have already discussed. Iron deficiency may be responsible for the production of anemia which results in glossodynia and atrophy of the tongue starting at the lateral border and progressing over the dorsum. This is most frequently a manifestation of pernicious anemia, but it is seen in severe iron deficiency anemia. Other specific dental manifestations are not seen in anemia, although poor response of periodontal tissues is often conjectured due to lowered oxygen tension of the tissue.

Numerous other disturbances in nutrition which are non-specific in character and unproven in significance could but will not be discussed here. Those already discussed are the most important in dentistry. Their discussion does not indicate that they are common findings, and we should not assume that all of our patients exhibit them to varying degrees. The ocurrence of primary dietary deficiencies are very rare in this part of the world today. Therefore, most of our patients need medical rather than dietary advice.

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MANIFESTATIONES ORAL DE CARENTIAS ENDOCRIN E NUTRITIONAL

Dr. chir. dent. Donald A. Kerr (M.S.)

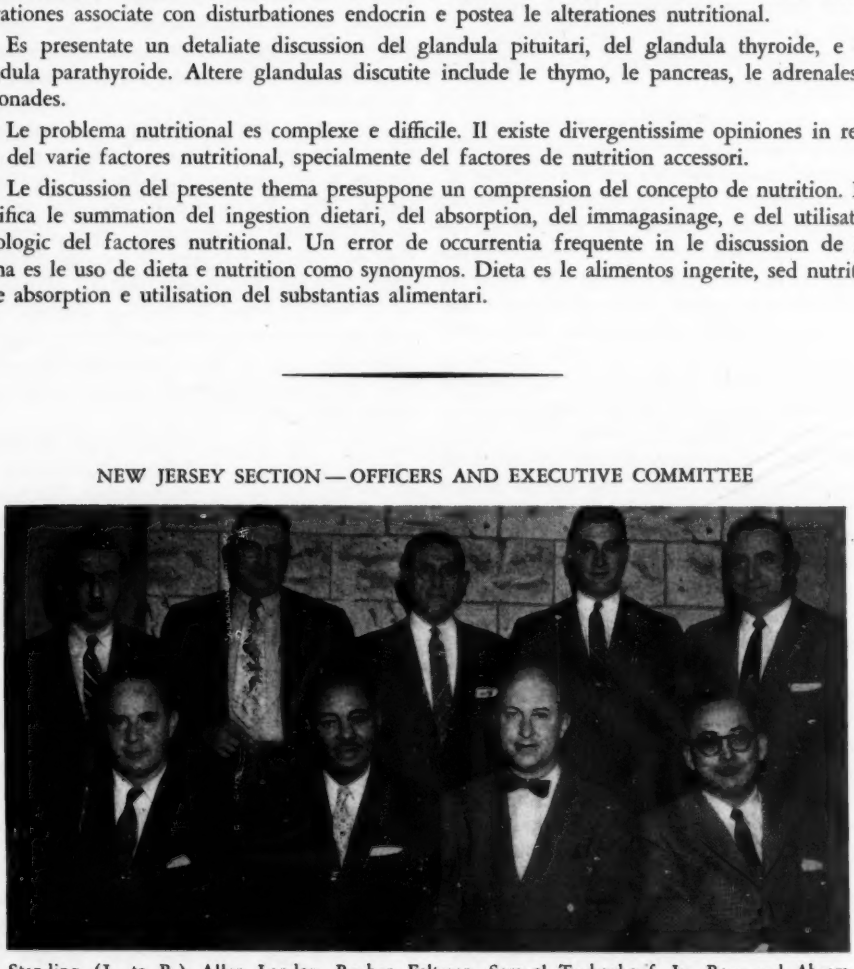
Le autor non desira applicar a humanos le constatationes experimental obtenite per ille in animales laboratorial. Le constatationes obtenite in animales sub inusualmente sever conditiones non permitte le supposition que illos existe in humanos sub minus sever conditiones. Le interpretation de constatationes in animales laboratorial como valide in humanos ha resultate in multe confusion in le litteratura non solmente medical sed etiam dental. Le autor discute alterationes associate con disturbationes endocrin e postea le alterationes nutritional.

Es presentate un detaliate discussion del glandula pituitari, del glandula thyroide, e del glandula parathyroide. Altere glandulas discutite include le thymo, le pancreas, le adrenales, e le gonades.

Le problema nutritional es complexe e difficile. Il existe divergentissime opiniones in re le rolo del varie factores nutritional, specialmente del factores de nutrition accessori.

Le discussion del presente thema presuppone un comprension del concepto de nutrition. Illo significa le summation del ingestion dietari, del absorption, del immagasinage, e del utilisation histologic del factores nutritional. Un error de occurrentia frequente in le discussion de iste thema es le uso de dieta e nutrition como synonymos. Dieta es le alimentos ingerite, sed nutrition es le absorption e utilisation del substantias alimentari.

NEW JERSEY SECTION — OFFICERS AND EXECUTIVE COMMITTEE



Standing (L. to R.) Allen London, Reuben Feltman, Samuel Turkenkopf, Ira Ross and Abram Chasens, Delegate N. J. Section. Seated (L. to R.) Morton DeScherer, Editor; Everett Simmons, President; Frank Schambach, President Elect; and Louis Kelsten, Sec'y-Treas.

NEW JERSEY SECTION WORKSHOP REPORT

Focal Infection With Relation to Dental Medicine

The membership of the New Jersey Section of the American Academy of Dental Medicine conducted its Second Annual Workshop on April 15, 1956 at the Hotel Douglas in Newark, New Jersey. The topic of the workshop was "Focal Infection with Relation to Dental Medicine."

This excellent report was prepared by Morton DeScherer, Editor of the New Jersey Section.

Dr. Paul Boyle, Professor of Oral Pathology at the University of Pennsylvania and President of the International Institute for Dental Research, opened the meeting as moderator of the workshop with many significant historical and scientific observations. His summations and conclusions are presented at the end of this report. During the meeting Dr. Boyle was honored wth the presentation of a placque "for distinct contributions to Dental Medicine." This was offered by Dr. Everett Simmons, President of the New Jersey Section, on behalf of the membership.

Each of five topics relating to focal infection had a discussion leader and a group of participants. Each group chose a secretary to record data. After the discussion period the secretaries read their data to the group at large and the discussion leaders formed a panel to answer questions and indulge in further observations.

Following are the topics, the discussion leaders and the secretaries whose summaries are herewith presented:

PRESENTATION OF PLAQUE TO DR. PAUL BOYLE, MODERATOR OF N. J. WORKSHOP

(L. to R.) Dr. Everett Simmons, President, N. J. Section; Dr. S. Leonard Rosenthal, Academy President; Dr. Paul Boyle, Recipient of Plaque; and Dr. Frank Schambach, Chairman of the Workshop. The citation on the plaque reads "For distinct contributions to Dental Medicine."

PERIODONTAL DISEASE

It was decided by this group that Focal Infection is to be reckoned with as a disease entity. Each member, as a result of clinical observation, felt that there was a direct relationship between a periodontal condition and an

PERIODONTIA—Allan N. Arvins, D.D.S. ..... Leader
Barney Polskin, D.D.S. ..... Secretary

INTERNAL MEDICINE—J. Reuben Budd, M.D. ..... Leader
Reuben Feltman, D.D.S. ..... Secretary

ENDODONTIA—I. B. Bender, D.D.S. ..... Leader
Harold A. Lentz, D.D.S. ..... Secretary

ORAL SURGERY—Martin S. Protzel, D.D.S. ..... Leader
Allan London, D.D.S. ..... Secretary

PATHOLOGY—George Stein, M.D., D.M.D. ..... Leader
Ira Ross, D.D.S. ..... Secretary

associated systemic problem, particularly in the field of ophthamology and cardiology. The thought occurred that a focus per se is insignificant when an isolated lesion, but body defenses vary with each individual and its importance varies accordingly.

There is apparently no definite evidence by laboratory procedures that involved teeth are not potential foci of infection. Conversely, many teeth are condemned when there is no evidence that they are involved.

It was further agreed that because of the larger surface area involved and easier route for extension, periodontally involved teeth were cause for greater concern than periapically involved teeth.

A "trigger area" on a previous base of lowered resistance resulting from one or a combination of conditioning factors seems to be the most usual picture. These conditioning factors are stress, nutritional deficiencies, blood dyscrasias, traumatic injury and pre-existing disease.

In conclusion, whether or not a focus is known to be causative, for the sake of the general body health and the dental condition itself, it must be treated and corrected.

If improvement results, so much the better.

To quote Hyman:

"Focal infection of dental origin is much less common than its enthusiasts believe and much more frequent than its skeptics allow."

INTERNAL MEDICINE AND FOCAL INFECTION

It is strongly suggested that all dental patients upon the first visit have a good medical history. This will give the dentist a lead as to any systemic disease and also be helpful in preventing unfortunate complications. From the internal medicine point of view, focal infection plays a role in the following conditions:

1. Arthritis. It is only of importance in mono-articular form of disease where one and perhaps two joints are involved. In this type of disease very often the removal of a focus may give dramatic results. Removal of foci of infection do not seem to play an important part in the other forms of arthritis such as rheumatoid arthritis, degenerative arthritis, gouty arthritis, etc.

2. Ocular disease such as uveitis, keratitis and corneal ulcer.

3. Removal of any focus is important when the general health of the patient may be affected by it.

PREVENTION OF SUBACUTE BACTERIAL ENDOCARDITIS

If a person gives a history of a previous rheumatic infection or has been treated for valvular heart disease or congenital heart disease, prophylactic penicillin should be given. The recommended doses is as follows: 600,000 units aqueous penicillin plus 600,000 units of procaine penicillin in oil, intramuscularly 30 minutes pre-operatively. It is advisable to be cautious before giving penicillin parenterally in a patient with allergy as occasionally fatal reactions may occur. Topical applications of penicillin ointments, lozenges and small cones of penicillin may produce hypersensitivity. If there is a question of doubt as to sensitivity, it is advisable to consult with the medical attendant on the case.

The subject of anti-biotics was discussed fully. The following phases were taken up.

1. Indications.
2. Resistance.
3. Complications.
4. Sensitivity tests.
5. Use of multiple anti-biotics.

Emphasis was placed on adherring to firm surgical principles.

STEROIDS

The importance of the alarm reaction and its effect upon the human organism was discussed It was emphasized that if a patient has been receiving steroid therapy and stops this form of therapy before a surgical procedure, serious complications may occur. It is advisable to discuss any such case receiving steroid therapy with the attending physician. The role of ACTH and cortisone in bleeding was discussed.

It was emphasized that when a physician refers a patient to a dentist, the dentist acts in the capacity of a consultant and should not

blindly remove teeth or do any procedure solely on the recommendation of the physician. Closer cooperation between physician and dentist is desirable.

ENDODONTIA AND FOCAL INFECTION

Focal infection was discussed as a biologic possibility that could produce systemic disease but its development is limited and doubtful. Only under certain conditions could this phenomen develop.

A distinction was made between a focus of infection and focal infection. A focus of infection was defined as any local inflammatory area containing bacteria that can potentially gain entry into the lymphatic and circulatory system. Focal infection was defined as the development of a metastatic lesion from a primary source of infection in a remote part of the body that had a pre-existing organic lesion.

Before focal infection can develop, certain criteria must usually be present.

1. A focus must be present;
2. Bacteria must be present in the focus;
3. The focus must be traumatized and hemorrhage ensue (bacteremia)
4. An organic lesion must be present in a remote part of the body.

There are other conditions under which focal infection can develop.

1. Acute infections, in which there is an increase in cell permeability because of the presence of certain enzymes, e.g., hyaluronidase.
2. Virulency of bacteria.
3. Tissue breakdown with ultimate entry of the bacteria into the blood stream.
4. Diapedesis. This is a relatively slow process and the organisms would be destroyed by phagocytosis.

It is more likely that under acute conditions, focal infection can develop.

There are certain natural defense mechanisms in the body that would prevent the development of focal infection:

1. The dilution effect of the blood neutralizes the bacteria and toxins that may enter the blood stream;
2. The blood is a liquid tissue that possesses natural antibacterial property;
3. Phagocytosis, present in the reticulo-endothelial system;
4. The formation of antibodies.

These barriers plus the absence of an organic lesion elsewhere in the body would tend to minimize the possibilities of development of focal infection.

The development of subacute bacterial endocarditis following extraction of teeth is a classic example of focal infection. Treatment and filling of a pulpal or periapical involved tooth is less apt to cause subacute bacterial endocarditis than extraction because the factor of trauma and hemorrhage with a resultant bacteremia would not be introduced.

In a vital extirpation of an infected pulp in a patient with a history of rheumatic heart disease, the tooth could be treated pre-operatively by sedation with zinc oxide and eugenol dressing to reduce the inflammation, virulency and number of bacteria. Prior to extirpation or entry into the root canal the patient should be pre-medicated with 400,000 units of penicillin (100,000 units of aqueous penicillin G and 300,000 units of procaine penicillin) and one gram of dihydrostreptomycin, one hour before manipulative procedure. In a limited number of cases of vital extirpation, bacteremias were demonstrated.

Areas of rarefaction at the apices of teeth are areas of reaction to irritation which may be chemical, mechanical or bacterial. A preoperative culture will determine whether we are dealing with an infective process. A distinction is therefore made between infection and inflammation. Sterile areas of rarefaction can become infected through anachoresis. In endodontics we are primarily concerned with the removal of the irritants from the confines of the root canal. The apical tissue will heal if the irritant is removed.

It has been demonstrated that if the root canal is rendered sterile, the periapical tissue is sterile.

Dr. Bender reported a collective study of 1733 cases in which cultures were taken. In 673 cases which had areas of rarefaction 33.8%

proved to be negative when bacteriologic cultures were taken. In the remaining 1060 cases, in which there were no areas of rarefaction, 53.4% were negative. Culture results of all 1733 cases with and without areas of rarefaction, exhibited negative cultures in 45.8% cases. Smaller areas of rarefaction manifested a slightly higher incidence of positive cultures than large areas. It is possible to have an area of rarefaction in a diseased vital pulp.

The criteria of success of endodontically treated teeth is the absence of pain or swelling and complete regeneration of bone and a normal periodontal membrance. If the bone does not appear to be completely filled in, upon x-ray, after one year, the area may be fibrotic, or else not all the irritant has been removed. To remove any doubt as to whether it be fibrotic tissue or residual infection a resection or curettage can be done.

A tooth that has been treated endodontically with complete regeneration of bone and an intact periodontal membrane should not be considered as a focus of infection.

There are no systemic contraindications for endodontic treatment of teeth. In fact, in some systemic conditions and under certain situations it is preferable to extraction. Examples are patients with a history of rheumatic heart disease, hemophelia, blood dyscrasias, and liver diseases. Patients that have been exposed to radiation therapy in the regions of the jaw are much safer to have endodontics than extraction.

There is no evidence to show that patients with no pulpless teeth are in better health than those with pulpless teeth.

ORAL SURGERY

Oral focal areas of infection can invade the system by dissemination of

1. Bacteria
2. Bacterial toxins
3. Foreign protein or allergies.

The attraction of blood-borne organisms or their products to traumatized or sensitized areas is known as anachoresis. The effects of previous trauma from disease or accident may result in areas having this tendency, therefore being more receptive to invasion from foci of infection.

Erythema multiforme is an example of a sensitized area affected by a focus of infection. Other attraction areas may result from myocardial damage of scarlet fever, sterile vegetative growths on the mitral valve, rheumatic heart, arthritic joints, kidney damage, etc. Even intact non-carious teeth that have been traumatized are subject to this action.

Removal of teeth with possible foci have been reported as improving or even curing some eye conditions. General health of children has been remarkably improved in many cases after correction of poor dental health.

It is recognized that oral foci of infection and its focal manifestations do exist. How often is still questionable. At present there is little laboratory aid that is capable of identifying this specific reaction.

Laboratory procedures, although valuable, have their limitations and too much reliance should not be placed on them. Clinical evaluation deserves more emphasis.

The influence of stress, nutrition and psychosomatic factors will alter the defense mechanisms causing variation in resistance to infection.

There are several procedures in use as aids in attempting to determine foci of infection.

1. Injection of a local anesthetic into a suspected area to produce exacerbation of symptoms elsewhere.
2. Ultra-short waves used for the same purpose.
3. The use of the E. S. R. (Erythrocyte Sedimentation rate) where a rapid increase in the sedimentation rate is obtained.

More recently an excellent test has been developed. This is called Reactive Protein antiserum test. It consists essentially of the extraction from the blood of a protein which is specific for the individual infection, and adding it to an anti-serum developed for the test. The protein reacting with the anti-serum in the form of a very fine but definite precipitation is indicative of a positive result. The relationship of the above tests could be of great value.

Toxins may be released from foci without evidence of the presence of organisms. A dental root cyst is usually bacterial in origin. It degen-

erates as it proliferates and it will usually present an inflammatory reaction. Toxins cannot be ruled out. It is also possible that supposedly sterile apical areas of non-vital teeth and root canal filled teeth showing no X-ray evidence could produce reaction elsewhere in the body.

Case history of areas at apices of teeth with filled root canals should be made to determine whether the condition is a healing stage of previous treatment. If there is X-ray evidence of healing and good bone re-formation we can be reasonably sure of non-infection.

Transient bacteremias following extraction of teeth must be guarded against as it may result in the development of sub-acute bacterial endocarditis.

A history of the patient before extraction is pertinent, and adequate pre-operative anti-biotic therapy where indicated will preclude the development of this disease.

Anti-biotics should be given parenterally and not orally. The use of cortisone is very good in acute infection. Where the adrenal cortex is not functioning properly, cortisone will not help. In these cases the combination of penicillin and gantricin is excellent.

The pendulum may be swinging too far in the direction opposite to our previous concept of focal infection with a resulting disregard of possible effects. Dentistry is a health service and each dentist should be cognizant of the fact that all oral foci of infection should be eliminated.

PATHOLOGY

The section discussion on pathology dealt with the investigation of microscopic aspects of periapical changes resulting from inflammation related to bacterial activity. It consisted of the showing of photomicrographs from Dr. Stein's collection, and discussion between the leader and participants.

The first phase dealt with the microscopic aspects of acute inflammation resulting in abscess formation in the periapical area. Changes noted were a widening of the periapical periodontal space with necrosis of the soft tissue elements and resorption of the alveolar bone. Replacement of the fatty marrow by fibrous marrow was discussed as the reparative reaction in the bone which had previously undergone inflammatory changes. It was further noted in the healed bone that, although the marrow had undergone fibrosis and thereby had lost its potential for hematopoesis, the surrounding bone trabeculae could undergo normal osteogenesis and osteoclasis. Therefore, it was felt that the rythym of bone desposition and resorption was restored, but that the special function of hematopoesis was not restored.

Following the discussion of periapical abscess production and sequelae, cyst formation was investigated. First, the important part played by the epithelial rests of Malassez in cyst wall production was reviewed. This included a discussion of the organization of periapical epithelium following pulp degeneration and infection. Cholesterol slits in the cyst lumen were described as the breakdown product of epithelium. It was felt that a cyst may be the end result of one of several different patterns of epithelial proliferation.

If a periapical abscess is present, the epithelium of the epithelial rests of Malassez may proliferate. Due to the tendency of the epithelium to cover surfaces, the abcess may be completely surrounded and lined by epithelium leading to the development of a cyst which is in open communication with the pulp canal.

In addition, cysts may be seen which are not in communication with the root canal. The source of this type of cyst is from the degeneration and liquifaction of cells inside a solid epithelial proliferation. An apical epithelial obstruction such as a cyst may not be completely sealed, and may act as a mesh through which active infection may pass. Epithelial obstruction and cyst formation can be considered an attempt of the body to heal periapical infection.

It is impossible to determine the true nature of an apical radiolucency without microscopic examination of the tissue. The three main varieties which may result from pulp degeneration and infection are acute and chronic alveolar abscess, apical granuloma, and periapical radicular cyst. These represent, respectively, occlusion of the apical foramen by varying degrees of inflammatory cells and organizing

mesenchymal tissue (fibrous capsule), and separation of the infected root canal from its surrounding invironment by the formation of an epithelial lined sac.

CLINICAL IMPORTANCE OF PERIAPICAL LESIONS

Not all non-vital teeth should be considered as foci of infection, but primarily those which show some evidence of some active inflammation. The three types of noxious materials which may emanate from such an inflammatory zone are bacteria, bacterial toxins, and chemical products of the inflammatory exudate such as leukotoxin, necrosin, and pyrexin.

CLINICAL SIGNS OF PERIAPICAL INFLAMMATION

1. Sensitivity of a tooth to percussion. (This presupposes that the pain in a given tooth is not the result of occlusal trauma.)
2. Sensitivity of the bone in the apical region to pressure.
3. Enlargement and tenderness of regional sub-maxillary lymph nodes.

If any of these three conditions are noted, it may be presumed that inflammation is occuring in the periapical area from which bacteria, bacterial toxins, or chemical by-products may emanate.

DISCUSSION

Recently two methods of evaluating possible foci of infection have appeared in the German literature. The first of these is by the injection of a product known as a "pyrifer." It is a suspension of certain dead apathogenic strains of B. coli. It is produced in three concentrations; one unit corresponds to 1,000,000 germs. Its purpose is to produce artificial fever. When a small dose, 5 units (5,000,000 germs), is injected intravenously, an exacerbation of periapical foci and of the secondary diseases can frequently be observed.

The second method involves the injection of an anesthetic, "Impletol," into the periapical area of the tooth. This injection produces the disappearance of pain symptoms in a distant site within seconds, if the injected tooth is the source of infection or infectious product causing the pain in the distant site. The effects last at least for eight hours. So far, these products have been used only experimentally, and the results of the experimentations are questionable. However, their use is being continued and they may be of value in future investigations of the problem of focal infection.

As an example of systemic disease thought to be related to a dental focus, Dr. Stein reported that periapical inflammation and abscess formation is seen as ferquently in rheumatic fever patients as is tonsillar inflammation. He went on to state that it has been noted that an acute exacerbation in either of these two sites usually precedes either the onset or a flare-up of rheumatic fever in about one to two weeks.

SUMMARY OF DISCUSSION

1. Three types of periapical lesions may result from pulp degeneration and infection; alveolar abscess (acute or chronic), dental granulama, and periapical cyst. The developmental stages and the microscopic characteristics of each were reviewed.
2. The clinical criteria for evaluating the presence of a focus of infection were suggested.
3. Experimental methods of evaluating whether a given infected area may be acting as a focus for changes in a distant site were discussed. The use of "Pyrifer" and "Impletol" were reviewed.
4. An example of a systemic disease, rheumatic fever, which may arise from a dental focus, was discussed together with the fact that a certain rythym seems to exist between the time of oral inflammation and the later joint symptoms.
5. This section recommended that studies be continued to gain additional information on the problem of focal infection. So much of the cause and effect relationship is still unknown that it is still premature to state either that such a relationship does or does not exist.

FOCAL INFECTION—SUMMARY

Paul E. Boyle, D.M.D.

Microorganisms of many varieties are present on the oral mucous membranes as on many body surfaces. When such organisms penetrate

into the tissues they may multiply and cause death of the host; they may be destroyed and eliminated; or a state of mutual toleration may be established. A focus of infection is defined as an area of tissue within which living microorganisms persist. Focal infection is damage to remote sites such as the joints, heart, kidney or other areas consequent upon dissemination of bacteria or their products from a focus of infection. Many well recognized diseases may show a local lesion from which bacteria or toxins may spread to affect other areas. Diptheria, typhoid fever, tuberculosis and syphilis demonstrate such a sequence. Metastatic abscesses in brain, kidney or elsewhere may have their origin in an abscess of the skin, about a tooth, or in other primary sites. Gonorrheal infection may be associated with arthritis. In rheumatic fever a previous upper respiratory infection with alpha strain, beta hemolytic streptococci is believed to sensitize the joints and other tissues so that after a characteristic latent period, the signs and symptoms of the disease are produced. Recurrences may be prevented by the use of antibiotics to control or prevent sore throats caused by such streptococci. Streptococcus viridans, a common inhabitant of the mouth and throat and often present in chronic inflammatory reactions about periapically diseased teeth, may lodge on heart valves causing sub-acute bacterial endocarditis, particularly where there are congenital malformations or scarring following rheumatic fever.

"Focal infection," as used by Billings, Rosenow, Price and a host of others in the years following 1910 after Hunter decried "oral sepsis," was advanced as a cause of many chronic illnesses. Symptom free teeth, tonsils, prostate glands, gall bladders, appendices and other tissues were surgically removed from many thousands of patients in an attempt to remove primary focus of infection. Extreme advocates of the theory of focal infection attributed mental illness, hypertension, cardiac and renal disease, peptic ulcer and other conditions to the dissemination of bacteria from foci about the teeth, tonsils or elsewhere.

Enormous numbers of teeth were extracted, tonsils removed and other operations performed in the hope that systemic illnesses of indefinite etiology would be cured. When one operation failed or gave only temporary or perhaps psychogenic relief other operations were recommended.

After the furor had run its course, the sober, second judgment of the people and of the medical and dental professions reasserted itself. It was belatedly recognized that relatively few patients had been significantly benefited and that many had been handicapped by the loss of teeth. The present concept is that disease about the teeth, as elsewhere, should be recognized and eliminated by adequate treatment. Successful treatment can often be accomplished without extraction of the teeth concerned. The dentist by training and experience is best qualified to determine what procedures are indicated.

Endodontic treatment of pulp involved teeth is unassociated with the temporary bacteremia which has been shown to result from the extraction of teeth. Sterilization of the root canal, root canal filling and normal bone healing, as indicated by roentgenograms and freedom from symptoms, is regarded as effective elimination of a possible focus of infection. Periodontal disease offers a relatively extensive area from which potentially pathogenic organisms or their products may gain entrance to the blood stream. Adequate treatment may restore health and thus eliminate such potential foci of infection.

In most instances, the defense forces of the body are adequate to control temporary bacteremias following tooth extraction or periodontal treatment. When heart valve damage exists, the possibility of subacute bacterial endocarditis may be guarded against by the use of antibiotics prior to and immediately following dental treatment.

Monarticular acute arthritis may be associated with acute or chronic infections about teeth, tonsils or the accessory nasal sinuses. Certain types of ocular disease are considered by some opthalmologists to be associated with dental infections. These rare and incompletely proven associations appear to be the solid residue of fact that remains from the frenzy that followed the acceptance of the theory of focal infection.

Seated (L. to R.) Dr. Joseph Barkley, Dr. Marie Salutsky, Dr. Charles Conat, Dr. Edgar James, Dr. Edwin Deer. Standing (L. to R.) Dr. Edward Freimuth, Dr. Horton Kimball, Dr. Walter Nieman, Dr. D. Chalmers Lyons, Dr. Max Hart, Dr. Harold Rattner. Dr. James Ladd, Committee Chairman, and Dr. Donald Simon are not in this photograph.

THE GAVEL CHANGES HANDS

Retiring President S. Leonard Rosenthal presents the gavel to William M. Greenhut, newly elected Academy President.

ACADEMY FELLOWSHIP RECIPIENT

Dr. George F. Clarke is presented with a Fellowship in the American Academy of Dental Medicine by Past President George A. Bruns.

The Academy ladies graciously pose for mbarking visits to many points of i nd the sur

AMERICAN ACAD NTAL
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June 21, 4, 195

1956 - 195 FFICERS

Seated (L. to R.) Dr. C. Ch e-Presid Greenhut, President; Dr. Marc Presid to R.) Dr. Irving Yudkoff, Edit Witkin, G.

pose for embarking on a bus tour which included points of interest and the surrounding area.

ACADEMY OF DENTAL MEDICINE
Tenth Meeting
Cadillac-Detroit, Mich.
June 21-24, 1956

1956 - 1957 OFFICERS

Dr. C. Chester, President-Elect; Dr. William M. Dr. Marcel Witkin, Secretary; Dr. George Ilkoff, Editor G.

Seated (L. to R.) Mrs. Charles Conat, Dr. Marie Salutsky, Mrs. D. Chalmers Lyons. Standing (L. to R.) Mrs. James Ladd, Mrs. Horton Kimball, Mrs. Edward Freimuth, Mrs. O. B. Aubin, Mrs. Joseph Barkley, Mrs. Walter Nieman.

PRESIDENTAL PLAQUE PRESENTATION

Incoming President William M. Greenhut presents the Distinguished Service Award to retiring President S. Leonard Rosenthal.

Dr. S. Leonard Rosenthal, retiring Academy President presents Honorary membership plaques in the American Academy of Dental Medicine to Dr. Edward C. Stafne (Left) and Dr. Donald A. Kerr (center) before two hundred members, wives and friends of the Academy.

IS THE EVIDENCE ON THE RADIOGRAPH RELIABLE?

J. C. ESELMAN, D.D.S., F.A.C.D., F.A.A.O.R.*

From the beginning of the use of X-rays in dentistry (1896) and continuing to the present day, the dental radiograph has been accused of revealing false evidence. However, no proof of these statements, except verbal, is submitted, and no one has been given a chance to defend the evidence presented on the radiograph.

The densities produced on a dental radiographic film by the use of X-rays are subject to physical, geometric and chemical laws which are constant.

PHYSICAL PROPERTIES OF X-RAYS

There are five basic physical properties of X-rays which are extremely important in dental radiography.

1. X-rays are invisible. This places them in the category of an unseen and silent killer. The dentist should be familiar with the methods of measuring X-ray radiation and should be concerned with the amount of radiation to himself and to the patient. The (R) Roentgen unit of measurement of dosage is the most accurate, and can be accomplished by using a dosemeter. The Coin test is acceptable for measuring radiation, but is not too accurate. There are two methods of protection to the operator, distance and barrier.

2. X-Rays travel in divergent straight lines. This creates a problem in operative radiography. The tube must always be aligned to the film in two planes, the vertical and the horizontal. It is possible for either angulation, if improperly used, to obscure evidence on the finished film. There are three acceptable operative technics; the bisecting angle, the mathematical and the right angle. Our inability to recognize inaccurate angulations is one of the most important causes of the loss of faith in the dental radiography. (Fig. 1.)

3. X-Rays are Differentially Absorbed by Matter. This is the physical property of X-rays which makes it possible to produce a black and white film, and to distinguish between the different densities on the film. Enamel and dentine both record as radiopaque on a diagnostic film and the distinction between them is readily visible. If this physical property of X-rays was not constant, the production of films for diagnostic purposes would be impossible.

The voltage and amperage applied to the tube terminals to generate X-rays can be used either as a constant or variable factor on the modern X-ray machines. Too much or an insufficient amount of either one, can be used to obliterate the evidence on the radiograph.

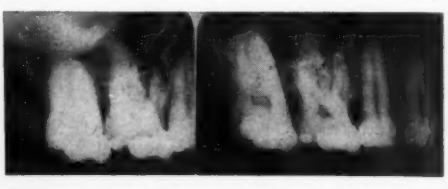

Fig. 1. On the left is an illustration of too high a vertical angulation which obscures the rarefaction over the buccal roots of the upper left first molar. On the right is the correction. The correction was accomplished by reducing the vertical angulation.

4. X-Rays Affect a Photographic Emulsion. X-rays have the property to affect the silver bromide salts in the emulsion in a manner similar to light. There is a constant physical relationship between the amount of X-ray reaction received by the film and the chemical processing of that film. That constant physical law is: "Those silver grams of the emulsion energized by the X-rays are acted upon and reduced to blackness by the developer in direct relationship to the intensity received from the X-rays. Those silver grams of the emulsion not acted upon by the X-rays or developer, are reduced by the fixing solution in relation to the intensity not received from the X-rays. The

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relationship between the time of exposure and processing of a film, can be utilized to reveal or hide the evidence on a dental radiograph. (Fig. 2.)

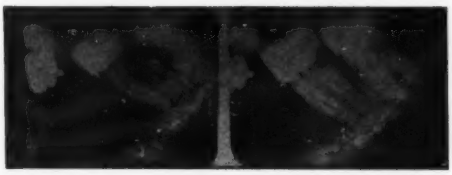

Fig. 2. An illustration of the relationship between the time of exposure and processing of a film being utilized to mislead the interpreter. The radiolucent area was interpreted as a cyst, however, due to early loss of the lower right first molar, the alveolar bone between the second bicuspid and second molar has atrophied and thus caused the unusual radiolucency.

5. X-Rays Cause Fluorescence. Salts of calcium tungstate fluoresce when acted upon by X-rays. This physical property is utilized to reduce the time of exposure. This physical property is a distinct advantage in extra-oral radiography and should be utilized.

Certain artefacts can be produced by careless handling of the film jacket. Unclean processing racks and the use of exhausted developer or fixing solutions are detrimental and will cause the evidence on the radiograph to be misleading.

LIMITATIONS OF THE DENTAL RADIOGRAPH

Other requirements which the dentist must absolutely possess are a knowledge of what the dental radiograph will reveal in relation to oral diagnosis and an expert knowledge of normal radiographic anatomy.

There are many limitations of the dental radiograph in relation to oral diagnosis. Here are nine of the more important ones.

1. The radiograph does not show infection, but the resulting influences of infection, trauma, chemical and surgical change. (Figs. 3, 4, and 5.)

2. The degree of intensity of the radiolucent area (that is the blackness) is not altered so much by its contents as by its anatomical location and the angulation used in taking the radiograph.

3. The radiograph does not tell whether the radiolucent area is sterile, infected, or in the process of repair. On occasions case history becomes very important. (Fig. 6.)

4. The radiograph does not show whether the radiolucent area contains fluid, pus, or granulation tissue.

5. The pulp is composed of soft tissue which does not record on the radiograph, therefore, vitality of pulp should be determined by thermal or electrical tests.

6. It is possible for a pulp to be hyperemic, inflammed, putrescent or gangrenous, with no visible apical change in the radiograph.

7. An acute abscess condition, no matter how much swelling is present in the soft tissues, will not be shown by the radiograph, unless bone destruction has previously occurred.

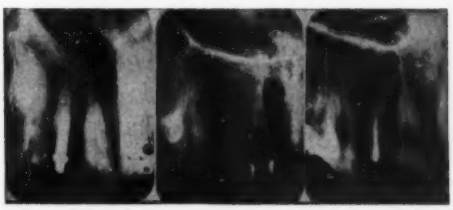

Fig. 3. Three upper left lateral incisors of different patients showing apical rarefactions. History reveals the three teeth were treated over five years ago. The logical deductions as to the cause of apical rarefactions would be infection at this time.

8. Any large radiolucent or radiopaque area in the maxillae will always appear smaller on the radiograph than its actual size (mesiodistally). This is due to the higher vertical angulations used in taking radiographs of the maxillae.

9. The radiograph does not show depths and localization methods should be used as indicated by the individual case. (Fig. 7.)

A knowledge of these limitations should impress dentists with the importance of using the evidence on the radiograph with the other methods of oral diagnosis.

The following is an axiom which will aid the dentist in interpretation of dental radiographs: "Usually the evidence on the radiograph is typical and characteristic of the particular con-

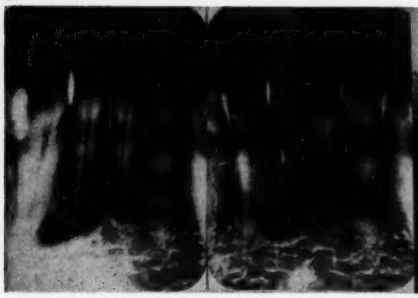

Fig. 4. Two radiographs of the same patient showing apical rarefaction of the lower left central incisor. However, this tooth was vital, had good color, and was free of objective or subjective symptoms. The logical deduction as to cause would be trauma. This condition is probably the forerunner of a cementoma.

Fig. 5. Three radiographs of the same patient showing an area of rarefaction. History revealed the patient had two apicoectomies trying to retain the upper left lateral, six years previously. The logical deducation as to the cause of the rarefaction would be surgery. Patient has no objective or subjective symptoms.

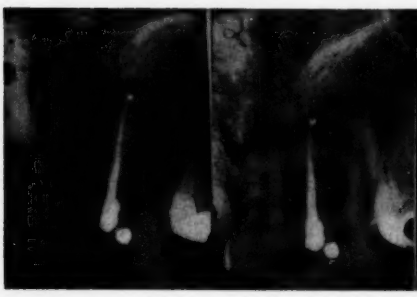

Fig. 6. Two radiographs of the same patient taken two months apart. In this case the interpreter would have to know which radiograph was taken first. Fortunately the radiograph on the right was taken last, showing some improvement.

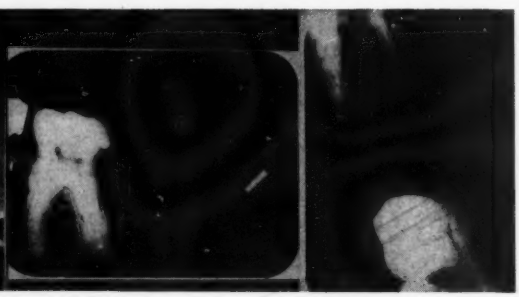

Fig. 7. A periapical and occlusal radiograph of the same patient. The interpretation concerns the cross-cut fissure bur which was broken off during the removal of the lower right third molar, ten years ago. The patient gave a history of having a surgical operation, five years later, and the approach was made from the buccal side in an attempt to remove the bur.

dition revealed. However, when radiographic and clinical evidence do not concur, then the information on the radiograph must be used as only one of the items in making a final diagnosis."

The dental radiograph does yield reliable information, but the degree of reliability must be determined by the dentist himself. In dentistry, the radiograph alone may establish the diagnosis but the history and clinical examination are absolutely requisite to the treatment. Radiographs may be deceptive, but they are not false.

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ES LE TESTIMONIO DE RADIOGRAMMAS FIDEL?

Dr. chir. dent. J. C. Eselman (F.A.C.D., F.A.A.O.R.)

SUMMARIO IN INTERLINGUA

Le radiogramma dental ha multe limitationes in su utilitate pro le diagnose oral. Es presentate un serie de nove del plus importante limitationes de ille genere.

1. Le radiogramma non revela infectiones sed le resultatos de infectiones, trauma, e alterationes chimic e chirurgic (Figs. 3, 4, e 5).

2. Le grado de intensitate del area radiolucente (i.e. le area nigre) non se altera tanto in consequentia de su contento como plus tosto in consequentia de su location anatomic e le angulation usate in obtener le radiogramma.

3. Le radiogramma non revela si le area radiolucente es sterile, inficite, o in stato restauratori. In certe situationes le historias del patientes deveni multo importante (Fig. 6).

4. Le radiogramma non revela si le area radiolucente contine fluido, pus, o histos de granulation.

5. Le pulpa es componite de histos molle que non se registra in le radiogramma. Per consequente le vitalitate del pulpa debe esser determinate per tests thermic o electric.

6. Il es possibile que le pulpa es hyperemic, inflammate, putrescente, o gangrenose sin que iste condition deveni visibile in ulle alteration apical in le radiogramma.

7. Un condition de abscesso acute, sin reguardo al grado de tumescentia in le histos molle, non se manifesta in le radiogramma, excepte si previe destruction de osso ha occurrite.

8. Omne extense area radiolucente o radio-opac in le maxillas presenta in le radiogramma dimensiones inferior al realitate (mesiodistal).

9. Le radiogramma non revela le dimension de profunditate, e methodos de localisation debe esser usate, indicate per le requirimentos del casos individual (Fig. 7).

Le familiarite con iste limitationes deberea servir a convincer le dentista del importantia del uso de datos radiographic in combination con le altere methodos de diagnose oral.

INTERNATIONAL ACADEMY OF ANESTHESIOLOGY

The next meeting of the International Academy of Anesthesiology will take place on Thursday evening, October 18th, 1956, at 8 P.M. in the Buffalo Room of the Hotel Statler, 33rd Street and Seventh Avenue, New York City.

Information concerning membership in the International Academy of Anesthesiology can be obtained from the Executive Secretary, Dr. Irwin G. Tomack, 355 East 149th Street, New York 55, N. Y.

RESORPTION OF DENTIN AND CEMENTUM IN TEETH

IRWIN WALTER SCOPP, B.S., D.D.S.*

Resorption of dentin and cementum in teeth is a unique disease. Unlike caries, it has been given scant attention in current dental literature. Too often, the initial lesion goes unrecognized in full mouth roentgenographic check up. Nevertheless, it becomes a perplexing entity when the disease is advanced and it is difficult to cope with in practice. It is variously referred to as idiopathic resorption, spontaneous intermittent resorption, sporadic resorption and metaplasia.

TYPES

Resorption in teeth may be either internal or external in type. Internal resorption is a disease in which the dentin of a tooth undergoes metamorphosis to a vascular granulation tissue devoid of odontoblasts. Related to this is external resorption, which dissolves the cementum first and then the dentin.

The dissolution of the dentin and cementum in teeth presents itself as a challenge to the dentist, since the etiology of the disease is obscure and the prognosis poor. In commenting on the etiology, Miller[1] states that, "possibly the change is predicated on an increase in local circulation producing a hyperemic decalcification such as occurs in bone."

OCCURRENCE

The paucity of cases of resorption appearing in the current literature would seem to indicate that such conditions are extremely rare; yet when one becomes aware of its entity and pathology, more and more cases are recognized and its appearance not uncommon. To quote Thoma,[2] "Idiopathic resorption of the tooth is seen more and more frequently. . . . It is more common than one would believe when looking for reports in the literature."

* Chief, Dental Service, Veterans Administration Hospital, New York 10, N. Y. Assistant Clinical Professor of Periodontia and Oral Medicine, New York University College of Dentistry.

HISTORICAL BACKGROUND

The early recorders of resorption in teeth were Bridgeman 1862,[3] Tomes 1872,[4] Gaskill 1894,[5] Fothergill 1899,[6] Mummery 1920,[7] Euler 1929,[8] Hopewell-Smith 1930.[9] These investigators generally attributed the phenomenon of resorption in a tooth to irritation and inflammation of one sort of another. More recently Cheyne and Berman 1947,[10] in discussing external resorption, considered it idiopathic.

LOSS OF VITALITY OF CEMENTUM THEORY

In 1931 Gottlieb and Orban[11] compared the external type with the change that occurs in replanted teeth in which the cementum undergoes resorption. They pointed out that in resorption the cementum of the tooth loses its vitality. It was their opinion that the mechanism of resorption centered around the loss of vitality of the cementum.

EXTERNAL ATTACK

The theory of external attack as the etiology was advanced by Euler and Meyer[12] in 1927. A case report was presented in which periodontal disease caused tooth resorption with channel

Fig. 1. Upper right second molar in a 22 year old female showing internal resorption of dentin from pulp wall. The entire pulp and root show vast areas of roentgenoluscency. The pulps of the adjacent teeth appear normal.

Fig. 2. 25 X enlargement of longitudinal section shown in Fig. 1. Note loss of dentin from pulpal wall and tremendous enlargement of pulp caused by osteolytic action.

formation. In 1934, Applebaum[13] reported cases purporting to show that an external opening causes resorption. These external openings may be through nutrient canals or accessory canals. In cutting the section, such openings may be easily missed. Supporting the theory that the process starts from without is a case reported by Aisenberg[14] in 1937. He demonstrated the presence of epithelium in a pulp that came from cell remmants (debris of Malassez of the periodental membrane.) In 1943 Thomas,[15] also found epithelium in the pulp.

MULTIPLE TOOTH INVOLVEMENT

The first case of multiple tooth involvement was reported by Soifer[16] in 1937; he cited internal resorption involving two upper incisors and a lower second bicuspid. In 1937, Munch[17] reported another case in which two upper incisors were involved. The fact that several teeth were involved simultaneously would seem to indicate

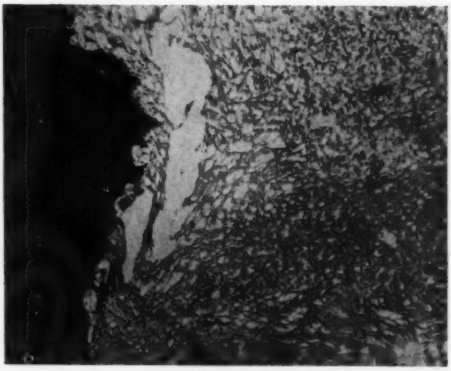

Fig.3. 100 X enlargement of same tooth. Note the very vascular granulomatous tissue and preponderance of inflammatory cells. The loose delicate connective tissue with odontoblasts found in normal pulp is absent. The lacunae along the dentin wall result from the osteoclastic activity.

that perhaps something systemic was the initiating cause.

PATHOGENESIS

Many theories, postulates and explanations have been offered to explain the phenomenon of resorption of dentin and cementum. Some of these are plausible, but not supported by evidence; others are untenable. The general conclusion one must draw, however, is that the pathogenesis of internal resorption is of idiopathic origin.

Bernier[18] refers to resorption of the tooth as Spontaneous Intermittent Resorption. He states, "Attempts to explain this phenomenon have not been entirely successful, because of the tendency to consider the tissue reaction as a basic disease process, rather than as a phase of the reactive maintenance pattern of calcified mesenchymal tissues. . . . It is likely that the defects are induced through a deficient maintenance pattern and therefore are formed without reference to identifiable causes."

DISCUSSION

Normally, the pulp is made up of a delicate, loose connective tissue with a lining or border of odontoblasts around the periphery. Around this is a straight housing of dentin. In inter-

Fig. 4. 30 year old male presented himself with a right central incisor which had a bluish discoloration. He stated that four days prior, the color of the tooth suddenly turned pink; and after three days, the color gradually changed to blue and the tooth darkened. There was no history of trauma. The enamel was easily penetrated with a bur. The material in the pulp was soft and caseous and had a foul odor. Treatment was root canal therapy and coronal amputation.

nal resorption, considerable changes occur. The odontoblasts are not present and instead a very vascular granulation tissue pervades the entire pulp chamber. This increased vascularization sustains and accelerates resorption. The pulp becomes increasingly larger at the expense of the dentin which is no longer straight and smooth. Instead, the dentin appears irregular as if eaten out, with many lacunae.

When the process of resorption reaches the enamel, a pink spot may occur. This is illustrated in Figure 4 and is caused by the highly vascular tissue and extravasation of red blood cells in the chamber being seen through the translucent enamel. The color changes to blue as the red blood cell undergoes degeneration. At this stage, the tooth may fracture easily under the biting stress. Paradoxically, the entire disease

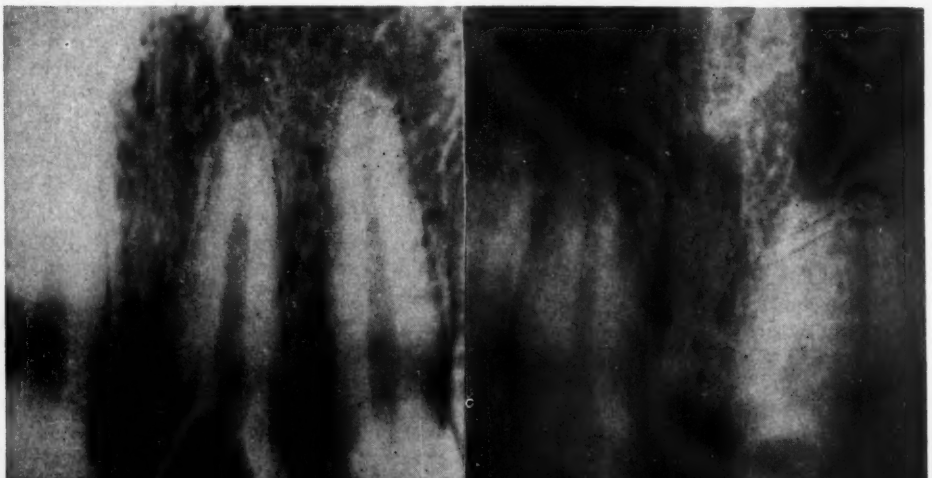

Fig. 5. Roentgenogram of right central incisor of case illustrated in Figure 4 shows internal resorption of the pulp. The coronal aspect of the pulp shows advanced resorption. Similarly, but less involved, is the coronal two-thirds of the root.

Fig. 6. Roentgenogram showing internal resorption and metaplasia of right central incisor in a thirty-four year old male. The pulp shows resorption of dentin but the root canal is completely filled in with a substance that roengenographically is equal in density to the dentin. Left central incisor has normal pulp.

Fig. 7. Roentgenogram of forty-one year old male with left central incisor showing internal resorption of the pulp and root canal, external resorption of the apex. The left central shows a physiologic filling-in of the root canal. The left lateral incisor appears normal.

Fig. 8. Roentgenogram showing external resorption of the left central incisor on the mesial aspect of the root. This is a case of early resorption.

process is generally asymptomatic which fact serves to underline the need of early recognition.

There are instances where resorption is started in the periodontal membrane. Here then is dissolution of the cementum and the dentin which finally reaches the pulp. This process is termed external or peripheral resorption. Figures 7 and 8 illustrate external or peripheral resorption. Bernier[2] relates internal and external resorption embryologically, "In odontogenesis, the ancestral cells of the pulp and periodontal membrane are similar mesenchymal forms having the potential to elaborate a matrix which can be calcified . . . therefore, the process may begin externally as well as internally, or simultaneously in both lesions. This need not be cause for wonder since these tissues have a basic similarity or potential."

In the external type of resorption, there is very often an attempt to repair by the deposition of an osteoid tissue, which is more radiolucent than dentin or cementum. The osteodentin and osteocementum are inadequate replacement for dentin and cementum and are a feeble attempt at repair.

In internal resorption too, an attempt at repair is constantly going on. In apposition to resorption, an osteoid tissue is laid down. Both processes go on simultaneously as in bone physiology. Since the newly laid down osteoid tissue is different than the normal pulpal tissue which it replaces, a form of metaplasia may result.

Often, secondary dentin will form apically and seal off the disease. This is a more successful attempt at repair and is illustrated in Figure 6. The entire root canal becomes filled with dentin.

TREATMENT

Where resorption has not progressed too far, root canal therapy is the treatment of choice. However, if the area of resorption is advanced or if it is so situated as to preclude root canal therapy, extraction of the tooth is indicated.

All this points to the need of early recognition of the lesion. Inasmuch as the disease process is asymptomatic until far advanced, it is important to detect resorption on routine full mouth roentgenographic series. In addition to checking full mouth series for caries, bone loss and faulty restoration, one should be prepared to recognize resorption in its early stages.

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RESORPTION DE DENTINA E CEMENTO IN DENTES

Dr. dent. chir. Irwin Walter Scopp (B.S.)

SUMMARIO IN INTERLINGUA

Resorption de dentina e cemento in dentes es un morbo unic. Per contrasto con carie, illo ha recipite pauc attention in le currente litteratura dental. Frequentemente le lesion initial escappa al observation in le complete roentgenographia del bucca. Tamen, illo deveni un entitate de grande perplexitate quando avantia, e alora illo es difficile a manipular in le practica. Le morbo es variemente designate como resorption idiopathic, spontanee resorption intermittente, resorption sporadic, e metaplasia.

Resorption dental pote esser de typo interne o de typo externe. Le resorption interne es un morbo in que le dentina de un dente suffre un metamorphose in vascular histos de granulation, disproviste de odontoblastos. In resorption externe, le cemento es dissolvite primo, le dentina subsequentemente.

Quando le resorption non es nimis avantiate, therapia de canal del radice es le tractamento de election. Tamen, quando le area de resorption es avantiate o si illo es locate de maniera que therapia de canal del radice non es possibile, le extraction del dente es indicate.

Omne iste considerationes signala le desirabilitate del prompte recognition de iste lesion. Viste le facto que le morbo es asymptomatic usque illo es multo avantiate, il es importante deteger resorption in routinari series roentgenographic del bucca integre. A parte le examine de series roentgenographic del bucca integre pro carie, perdita de osso, e restaurationes incorrecte, on deberea etiam interessar se al detection de resorption in su stadios precoce.

THE RELATION BETWEEN RAMPANT BUCCAL (SMOOTH SURFACE) DENTAL CARIES, ORAL BACTERIAL FLORA, AND SYSTEMIC DISEASE*

DON CHALMERS LYONS, D.D.S., M.S., PH.D.

A review of dental literature discloses the fact that seldom is there any mention of smooth surface caries as an identity in the determination of caries indices, but the type has become very significant in dental research.

Until recently only caries of occlusal grooves, fissures, and interproximal areas were produced experimentally in animals. Weisberger et al[21] in 1940 did report some smooth surface decalcification in the molar teeth of rats from which the salivary glands had been removed. Stephen et al[17] was able to produce this type caries in the rat fed a low fat, high sugar diet (32% skim milk powder, 66% cane sugar, 2% whole liver substance). Folk and McClure[7] used diets containing 35% skim milk powder prepared by the spray and roller process and 25% dry whey powder and report a 50% incidence of buccal caries. Addition of the chelating agent ethylenediamine-tetraacetic acid which is able to withdraw calcium in an alkaline environment, to the diet, produces a great increase in number and extent of the caries, according to Stephan and Harris.[18]

The ability to produce this type of caries has stimulated research on the problem, particularly in the relation which the salivary secretions play to caries indices. The broad spectrum antibiotics control smooth surface caries at least initially. Carter et al[1] checked the role of oral phosphatases in human saliva and came to the conclusion that they are partly of bacterial cell origin and partly of glandular, but felt that the slight increases noted in caries active mouths were not sufficient to warrant any association conclusion. Muhler and Shafer[12] find that the feeding of dessicated thyroid with thiouracil resulted in an increase in caries, and when thiouracil is combined with fluorine, the fluorine is ineffective. Swartz and Shaw[19] find that the serous salivary glands exert the greatest protective action. Volker et al[20] noted that in hamsters there are more carious teeth when the saliva is on the alkaline side. Weisberger[21] and others have found that either abnormally diminished salivation or removal of salivary glands produce a high incidence of buccal caries. Lyons[11] reported that individuals with repeated multiple caries were in a high percentage, taste blind to phenolthiocarbamide. Others have reported a relationship with such systemic diseases as tuberculosis, glandular insufficiency, hypovitaminosis A, and nutritional imbalances. Hecht and Freidman[10] found that there are 58 times as many cervical caries in the habitual narcotic user as in their control group.

Fig. 1. D.·G. Female child age 6. Nutritional imbalance. Generalized vitamin deficiency.

In this study a check was first made to determine the incidence of buccal caries in normal patients. Examination of 500 nonselected patients regardless of sex, age, or race as they presented themselves for treatment disclosed the need for 1782 dental fillings. There were 693 disto-distal occlusal, 554 mesio-mesial occlusal, 298 occlusal, 203 buccal, 31 lingual, and 3 palatal. Total smooth surface cavities 237 or 13.2%, buccal only 11.4%. In this group only 6 needed more than 5 buccal fillings at the time

*Presented at the 56th General Meeting of the Society of American Bacteriologists, Houston, Texas, April 29-May 3, 1956.

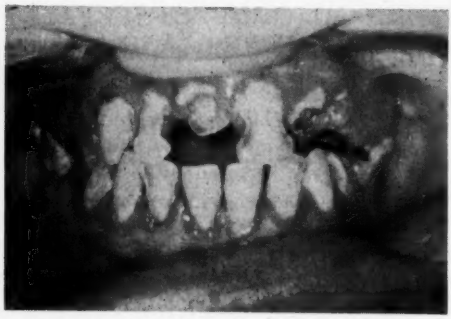

Fig. 2. M. M. Adult male. History of Diabetes.

Fig. 4. D. G. Adult male. History of tuberculosis now arrested.

of examination, the average being 2 as compared to the general average of 3.6. Therefore it would seem that in the normal dental patient without clinical complaint of illness that the smooth surface cavity is a small part of the dental problem, and that when they are massive and multiple one might expect some accountable salivary or systemic upsets.

A group of 11 cases have been accumulated in which extensive buccal caries have progressed to the point when total extraction was necessary. A complete medical-dental history was obtained from each patient. A total of 194 teeth were involved. Before extraction each tooth was isolated and cultures obtained from the surface of the carious area and after removal of the gross decayed material with a spoon elevator. A pooled salivary specimen was obtained from each patient.

The carious material was cultured in Synder test agar (bacto-tryptose, dextrose, sodium chloride, agar, and brom-cresol green), and incubated 24 to 72 hours. Caries activity was based on growth in this period and acid production according to color change of the media.

In only one of the eleven cases was there a 4+ acid production and marked caries activity. There was moderate activity in 8 and slight in 2 cases. Acid production corresponded.

CHART 1.

Number of teeth cultured				194	
Number of cultures obtained				388	
Acid production	0	1+	2+	3+	4+
Growth in 24 hours				4	18
" " 48 "		72	191	9	
" " 72 "		9	74	6	
No growth	5				

The results obtained in this small group seem to indicate that neither caries activity nor rate of acid development alone are the cause of the total tooth destruction noted. Strain differences in oral bacteria or the possibility of a species of bacteria of greater than normal destructive capacity was considered but none of our tests seem to indicate such.

In six cases there was a definite history of nutritional imbalance as evidenced in the two children by rate of growth, weight, height, and history of their average diet. Gum tissues and lips gave evidence of vitamin deficiency. Four adults had a history of progressive weight loss associated with, respectively, stomach ulcer, colitis, diabetes, and nephritis. In two cases there was glandular insufficiency as indicated by hypothyroid deficiency and thyroid adenoma, and the

Fig. 3. A. F. Adult male. History of chronic Colitis.

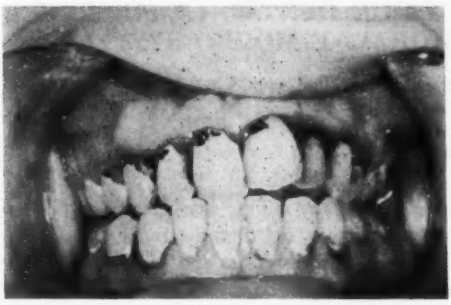

Fig. 5. D. Z. Adult female. History of parathyroid deficiency.

Fig. 6. G. M. Adult male. Blood deficiency. Abnormal clotting and prothrombin time. Excessively long bleeding time.

other a parathyroid deficiency. There was one case of tuberculosis (arrested and recently discharged from sanitarium at time of examination), and in one a blood deficiency with low hemoglobin, diminishing red blood cell count, prolonged bleeding (12 minutes), abnormal clotting and prothrombin time.

In these cases there was (1) breakdown of the usual resistance of the teeth to caries activity, (2) an apparent increase in bacterial growth factors without increase in rate of acid production, however there was an overall greater accumulative destructive action, (3) a breakdown in systemic resistance due to the presence of debilitating diseases, (4) a change in salivary activity as indicated by taste blindness to phenol-thiocarbamide, (5) development of poor dental hygiene in several cases.

No conclusions are drawn from this small group of cases except to note an apparent interrelation of salivary mechanisms. A more complete method of salivary secretion assay should be developed and further studies are needed into the relationship between enamel surface resistance and oral factors other than the bacterial flora.

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LE RELATION INTER RAMPANTE CARIE DENTAL (A SUPERFICIE LISIE), LE FLORA BACTERIAL ORAL, E MORBO SYSTEMIC

Dr. chir. dent. e phil. Don Chalmers Lyons (M.S.)

SUMMARIO IN INTERLINGUA

Le resultatos obtenite in iste parve gruppo de casos pare indicar que ni le activitate de carie ni le grado del disveloppamento de acido per se es le causa del total destruction dental que esseva notate. Differentias racial del bacterios oral o le possibilitate de un specie de bacterios con plus que normal capacitates destructive esseva considerate, sed nulle del tests empleate per nos pareva supportar un tal interpretation.

In iste casos il habeva (1) collapso del usual resistentia del dentes al activitate de carie, (2) un apparente augmento del factores de crescentia bacterial sin augmento in le volumine del production de acido (ben que con augmento cumulative del action destructive in general), (3) reduction del resistentia systemic causate per le presentia de un morbo invalidante, (4) alteration del activitate salivari, indicate per non-reaction gustatori a phenol-thiocarbamido, e (5) disveloppamento de inadequate hygiene dental in plure casos.

Nulle conclusiones pote esser derivate ab iste parve gruppo de casos, sed nos debe signalar le apparente interrelation con le mechanismo salivari. Deberea esser disveloppate un plus complete methodo de essayar le secretion salivari, e studios additional es requirite in re le relation inter le resistentia del superficie de emaille e factores oral altere que le flora bacterial.

SETON HALL COLLEGE OF DENTISTRY

The Seton Hall College of Dentistry will sponsor a postgraduate course in "Maxillo-facial Anatomy With Practical Applications" by Professor Harry H. Shapiro. The course will be given one morning each week for five weeks, October 3, 10, 17, 24, and 31, 1956. This course will be repeated in November and December 1956 and in January, February, March and April 1957. Applications must be received not later than the 15th of the preceding month. Tuition is $50.00.

For further information and an application contact Seton Hall College of Dentistry, Jersey City Medical Center, Jersey City 4, New Jersey.

Medical Views on Oral Problems

PARALYSIS FOLLOWING PENICILLIN INJECTION*

TO THE EDITOR:—In Queries and Minor Notes, in THE JOURNAL, March 10, 1956, page 921, is a question regarding flaccid paralysis of a leg occurring within 24 hours after intramuscular injection of penicillin on that side. This occurrence is almost certain to be due to the penicillin having been injected in close proximity to the sciatic nerve. Chances of full recovery within a period of six weeks to six months are quite good. Pain in the distribution of the sciatic nerve during the phase of recovery may occur. Physical therapy consisting of passive exercises or possible splinting of the ankle should be done in the meantime to prevent deformities.

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University of Utah
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* Queries and Minor Notes, J.A.M.A., June 9, 1956, p. 568.

BEHCET'S SYNDROME*

TO THE EDITOR:—*Where can a complement-fixation test on Behçet's syndrome (recurrent ulceration of the genitals, lesions of the mouth, and uveitis or iridocyclitis) be performed? I am under the impression that this disease is frequently overlooked.*

M.D., New York.

ANSWER.—Complement-fixation tests are based on the antibody response to an infectious agent. Behçet's syndrome has not been proved to be an infectious disease, and complement-fixation tests, therefore, are not used as a diagnostic measure in this disorder. At present, the diagnosis of the disease is made on the basis of recurrent attacks of oral aphthae, genital ulcerations, uveitis, and hypopyon, erythema nodosum, cutaneous ulcerations, arthritis, phlebitis, and central nervous system disturbances. Not all of these signs may be present in a given patient. The disease is resistant to all therapy. It has indeed been frequently overlooked, and this often seems to have been due to the fact that specialists tend to ignore recurrent signs in body areas remote from their own fields of interest. Recently Sezer (*Am. J. Ophth.* 41:41-55 [Jan.] 1956) elaborated on the etiological role of a virus in this disease and the result of the complement-fixation tests of his patients in Turkey. These findings have not been confirmed to date.

* Queries and Minor Notes, J.A.M.A., June 16, 1956, p. 668.

STERILIZATION METHODS FOR SMALL OFFICE*

TO THE EDITOR:—*1. Which form of sterilization is preferred in a small general office—dry heat (oven) or autoclave with steam under pressure? 2. Which form of sterilization best preserves the cutting edges of knives and scissors, rubber goods, and cotton goods?*

Irving Saffran, M.D., Mattapan, Mass.

ANSWER.—Steam under pressure, provided by an autoclave, is preferred for the sterilization of most items. It has the following advantages: 1. Lower temperatures are required, and therefore the damage to rubber goods and fabrics is less than that caused by the higher temperatures necessary for hot-air (oven) sterilization. 2. Shorter sterilization cycles are required for autoclave sterilization. Cutting edges of knives and scissors are best preserved by sterilization with hot air. Instructions for required time-temperature cycles for packs, gloves, and instruments, as well as for preparation of items for sterilization are supplied by manufacturers of sterilization equipment.

* Queries and Minor Notes, J.A.M.A., June 9, 1956, p. 566.

GRANULOMATOUS UVEITIS*

TO THE EDITOR:—*What role do dental foci, such as apical abscesses, play in the etiology of a severe granulomatous unveitis?*

M.D., West Virginia.

ANSWER.—Generally, dental foci or any other focal infections are not considered to have an etiological role in granulomatous uveitis. Syphilis, tuberculosis, sarcoidosis, toxoplasmosis, brucellosis, sympathetic ophthalmia, fungi, and viruses are the usual causative agents. Nongranulomatous uveitis may in some patients be associated with a septic focus in the teeth, tonsils, nasal sinuses, genitourinary tracts, or intestinal tracts. The underlying fundamental cause in this type of inflammation is a bacterial allergy particularly due to streptococci. Irrespective of the possible role of focal infection in nongranulomatous inflammation, good hygiene requires the elimination of easily accessible conditions such as apical abscesses.

* Queries and Minor Notes, J.A.M.A., June 16, 1956, p. 669.

ORAL MUCOSAL SMEARS IN DETECTION OF GENETIC SEX†*

In the nuclei of most cells from females there is

found a distinct mass of chromatin that corresponds to the sex chromosome. It is genetically determined, has been observed in cells from every type of female human tissue studied, and is very seldom found in the nuclei in males. These facts were verified in a study of 125 normal people of various ages by obtaining scrapings from the inner surface of the cheek with the edge of a tongue depressor .The scrapings, fixed and stained for microscopic examination, made it possible to tell the sex of the donor correctly in every normal case.

The test was also applied to three female pseudohermaphrodites, one male pseudohermaphrodite, and one enuchoid female with gonadal dysgenesis. In these five instances the gentic sex agreed with the gonadal sex. The test yields valuable information, and for this purpose the Papanicolaou technique proved very useful, although the Papanicolaou stain was less satisfactory than cresyl violet.

* Summary, J.A.M.A., June 23, 1956, p. 683.
† by Robert B. Greenblatt, M.D., Oscar Mateo de Acosta, M.D., Efrain Vázquez, M.D. and Dewitt F. Mullins Jr., M.D., Augusta, Ga.

EYE, EAR, AND TEETH DEFECTS IN YOUNG GIRL*

TO THE EDITOR:—*A 7-year-old girl at birth was found to have congenital glaucoma, which has been controlled medically. When 6 years of age, she developed bilateral cataracts, which were removed a few months ago, resulting in fair vision. The child has always had poor teeth. The first tooth erupted when the child was 6 months old, but, by the time she was 15 months old, most of the deciduous teeth were destroyed by caries. Her permanent teeth are now very carious. At the age of 6 she developed moderate deafness. Audiometer testing revealed a loss of high tones due to nerve damage. The child has always had dry skin, very dry hair, and brittle nails. There is no loss of sweating. Blood glucose and calcium levels, complete blood cell count, and urine were all within normal limits. Bone age of hands and wrists was normal last year. The child has no other known anomalies. She is alert and intelligent and does well in school. Her present height is 4 ft. (121.9 cm.) (3rd percentile) and her weight is 45 lb. (20.4 kg.) (15th percentile). The child had measles at the age of 2, and chickenpox at the age of 3; both were of a mild nature. There have been no other illnesses. There is no history of rubella during the mother's pregnancy. Could there be a common etiology to these conditions?*

C. Herbert Crane, M.D., Livingston, N. J.

This inquiry was referred to two consultants, whose respective replies follow.—ED.

ANSWER.—Several conditions might be considered to explain the defects in this child, but it seems apparent that rubella in the mother during pregnancy is not responsible. This conclusion is not based merely on a negative history for that disease, since it has been reported that rubella can occur without an exanthem. The defects that are present and the time that some of them developed are not consistent with the usual abnormalities that are attributable to intrauterine infection from rubella. On the basis of the history, it seems most likely that this is a case of congenital ectodermal dysplasia. The dry skin, dry hair, and defective teeth are characteristic of that condition. Two types have been reported: (1) anhidrotic, where the sweat glands are absent, and (2) cases where sweat glands are present. Eye defects are not always observed but have been mentioned by some authors. James (*Acta. paediat.* 41:229, 1952) states that ectodermal dysplasia even in fully developed states does not necessarily follow a fixed pattern. Generally, but not always, there seems to be evidence that the condition is inherited. It occurs more often in males than females. There is no cure.

ANSWER.—The above-described case fits the description of two syndromes that are characterized by the important features described. "Werner's syndrome (Ormsby, O. S., and Montgomery, H.: Diseases of the Skin, ed. 8, Philadelphia, Lea & Febiger, 1954, p. 694), progeria, characterized by shortness of stature, cavities, premature baldness, cataracts, hypogonadism, tendency to diabetes, calcification of the blood vessels, osteoporosis and metastatic calcification, is a heretofamiliar disorder tending to occur in brothers and sisters." Rothmund's syndrome (Rothmund, A.: Ueber Cataracten in Verbindung mit einer eigen thumlichen Hautgegeneration, von Graefes, *Arch. Ophth.* 14:159, 1868) is also a recessive hereditary disorder, beginning in early childhood. The shortness of stature and the changes in the muscles are not as marked as they are in Werner's syndrome, and the sexual development usually is normal. Cataract occurs in both syndromes. Osteoporosis is not observed in Rothmund's syndrome, and the skin has brownish pigmentation and telangiectasis associated with it. Rothmund's syndrome may occur singly or in siblings. The onset of cataracts is later than in Werner's syndrome. As a rule, sexual organs are not abnormal. Children are alert, intelligent, and socially at their chronological age level. As the disease is a heredofamilial one, treatment does not materially alter the course of development of the affected children. Results of operations for cataract in reported cases have been fair. Both syndromes may occur in incomplete form. The case described above seems to fit better into the Rothmund syndrome category.

* Queries and Minor Notes, J.A.M.A., June 9, 1956, p. 567.

CIRCUMORAL HYPERPIGMENTATION*

TO THE EDITOR.—*A 40-year-old Chinese woman began showing areas of increased pigmentation cir-*

cumorally and over the bony prominences. Blood pressure has remained normal, and the Kepler test shows normal results. Melanuria is present. Physical examination is negative, but the patient is very thin (94 lb. [42.6 kg.]—4 ft. 11 in. [149.9 cm.]), although her appetite is excellent. She has a temporomandibular arthritis that has respondent to roentgen therapy. She has multiple vague complaints and is quite distressed by the cosmetic problems posed. A chronic otitis, which has responded to therapy and antibiotics, is present. Pelvic examination is negative. In the past four months the pigmentation has increased in density until now it is almost black over the bony prominences, with some discoloration of mucous membranes. Are there any further diagnostic studies or any type of therapy that would be of avail?

Frank E. Glaster, M.D., Aiea, Oahu, Hawaii.

This inquiry was referred to two consultants, whose respective replies follow.—ED.

ANSWER.—Hyperpigmentation is usually attributed to physical or chemical agents, systemic abnormalities (particularly endocrine disorders), or cutaneous diseases. Circumoral hyperpigmentation occasionally occurs in patients who receive bismuth. It occurs, too, in erythrose peribuccale pigmentaire, a condition noted occasionally at the time of menopause in women with seborrheic skins. If a true melanuria exists, the presence somewhere of a melanoma should be suspected. If the pigment is due to hemogentisic acid or alkaptonuria, one of the inborn errors of metabolism such as ochronosis must be considered. The Puetz-Jaeghers syndrome of mucous membrane pigmentation associated with intestinal polyposis, Hodgkin's disease, or other lymphoblastomas may also manifest peculiar types of pigmentary disorder.

ANSWER.—Circumoral pigmentation and pigmentation over the bony prominences is seen in many conditions such as malaria, leprosy, pellagra, hemochromatosis, lymphadenoma, abdominal carcinosis, ochronosis, colonic polyposis, Riehl's melanosis, melanoleukoderma, and fixed eruptions from drugs. This reply is limited in its scope to pigmentation due to abnormal production of melanin, as attention was focused on melanuria. From the brief description given of the lesions and the fact that a biopsy of the hyperpigmented areas was presumably not made, it is difficult to make a definite diagnosis; however, the following diagnoses must be considered: adrenal cortical hypofunction (Addison's disease), melanoderma (chloasma), leukoderma, and ochronosis. Hypoadrenocorticism in a milder (subclinical) state than the well-known textbook description of it is much more common than is generally supposed. The multiple vague complaints mentioned and the size of the patient, despite her excellent appetite, could possibly be due to hypoadrenocorticism. The following tests will aid in the diagnosis of a deficiency state of the adrenal cortex: (1) serum sodium and potassium, (2) blood sugar, and (3) nonprotein nitrogen; also, the use of adrenocortical hormone for a short period of time may influence the degree of hyperpigmentation and thereby aid in the diagnosis. While the hyperpigmentation of adrenal cortical hypofunction is usually more diffuse and generalized, it can be localized to areas of normally increased hyperpigmentation. Until a definite diagnosis is made, treatment with a diet high in sodium and low in potassium and adrenocortical hormone is suggested.

* Queries and Minor Notes, J.A.M.A., June 16, 1956, p. 667.

RESPECTABLE HYPNOSIS*†

Asher is of the opinion that the sinister notoriety with which sensational literature has endowed hypnosis has driven away many who might make good use of it. He makes a plea that it be recognized as a reasonable form of treatment in certain selected cases and that it become more respectable. He does not advocate that it be used extensively nor that physicians designate themselves as "hypnotists" or that they specialize in the subject. Hypnosis is an adjunct to treatment; it does not replace other treatments, nor does it obviate the need for examination and investigations. The powerful effect of suggestion is generally realized; a new medicine may achieve very good results by suggestion. Hypnosis means using the suggestion in pure form, without the medicine. The author has used hypnosis as an ancillary method for the last 12 years, and for the last 6 years he has devoted one outpatient session a week to it. Hypnosis is a state of exaggerated suggestibility produced by suggestion and by fixing the attention.

The author generally induces the first hypnosis in a quiet, dark room, with the patient on a comfortable couch. He asks the patient to look at a light, usually an ophthalmoscope bulb. The reason for the dark room and the light is that if there is nothing else to look at it is easier to concentrate on the one visible thing. Suggestions are made repeatedly. At the first attendance only light hypnosis may be induced, but with subsequent sessions a deeper state may be obtained each time. Certain people never go very deep, but the effect of suggestion made under light hypnosis is often nearly as good as that made under deep hypnosis. In a good subject, after the first induction it is convenient at subsequent sessions to ask him to look at some object, such as the physician's finger or a paper-knife, in broad daylight. The less ritualistic and the more straightforward the procedure the better. The patient's ideas about hypnosis seem to play little part in its production, and some patients on waking say, "Is that some sort of gas you've got in that lamp?" and have no idea that hypnosis has been used. Results are described that indicate that hypnosis may have an effect on skin diseases, including warts, excessive sweating, alopecia, and eczema. The eczema-asthma syndrome of childhood and dermatoses made worse by scratching seem spe-

cially amenable. It may be helpful, too, in psychological conditions such as hysteria, anxiety states, and obsessional neuroses.

* Medical Literature Abstracts, J.A.M.A., June 2, 1956, p. 483.

† By R. Asher. Brit. M. J. 1:308-313 (Feb. 11) 1956 [London, England].

STILBAMIDINE AND TIC DOULOUREUX*

TO THE EDITOR:—Some statements about the use of stilbamidine in the treatment of tic douloreux made by Dr. Robert Dean Woolsey in the Dec. 31, 1955, issue of THE JOURNAL, page 1713, deserve comment. Since these statements are incongruous with established experience, that particular portion of the report is quoted: "Stilbamidine has recently been reported by Smith and Miller as having a beneficial effect upon the course of trigeminal neuralgia. Nearly every patient with trigeminal neuralgia who has entered my office recently has carried a clipping from the local newspaper or a recent copy of *Time* describing the wonders of this form of therapy. On reading the article, one finds that the drug was given over a period of one week and then discontinued. The pain usually subsided about a month later. Had sterile water been used, one could predict approximately the same results. Trigeminal neuralgia commonly comes and goes, and *post hoc, ergo proter hoc* reasoning has led to marvelous cures being ascribed to a great many drugs." The late chronic toxic effect of stilbamidine is confined to an unusual delayed neuropathy. The progressive sensory changes of paresthesia, hypalgesia, and anesthesia, usually confined to the face, are noted about two to five months after the administration of a course of stilbamidine. Napier and Sen Gupta (A Peculiar Neurological Sequel to Administration of 4:4' Diamidino-Diphenyl-Ethylene (M. & B. 744), *Indian M. Gaz.* 77:71-74 [Feb.] 1942) found a subjective disturbance of sensation over various distributions of the fifth cranial nerves. They suggested that the lesion was in the sensory nucleus of the fifth cranial nerve in the pons. Collard and Nevin (Affection of the Trigeminal Nerve Nucleus and Central Gray Matter of the Spinal Cord Following the Administration of Stilbamidine, *Proc. Roy. Soc. Med.* 40:87-88 [Nov. 7] 1946) also considered the lesion to be in the nucleus of the fifth nerve and, in addition, in the descending nucleus. The mechanism of the neuropathy is not understood. Oastler and Fidler (Cerebral Lesions Produced in Healthy Dogs by the Intravenous Injection of 4:4' Diamidino Stilbene, *Tr. Roy. Soc. Trop. Med. & Hyg.* 39-533-538 [June] 1946) administered fresh solutions of stilbamidine to 10 dogs and concluded that the neuropathic action of stilbamidine was within the central nervous system.

In our initial report, which Dr. Woolsey quotes incorrectly, it was suggested that the substitution of a hypesthesia for the sharp pain of tic douloureux by the use of stilbamidine might be a significant advance in the treatment of this painful disease and that stilbamidine might offer palliation in elderly patients in whom operation was contraindicated. The result obtained in this first patient, an 82-year-old woman, was encouraging. A total of 16 patients, so treated by us, was reported later, and excellent results were obtained in 15 patients and good results in one (The Treatment of Tic Douloureux with Stilbamidine, *Bull. Johns Hopkins Hosp.* 96:146-149 [April] 1955). Woodhall and Odom (Stilbamidine Isethionate Therapy of Tic Douloureux, *J. Neurosurg.* 12:495-500 [Sept.] 1955) have also reported good results following such treatment. A recent review of an increased number of our own patients with tic douloureux indicates the continued usefulness of stilbamidine. The plan of treatment has not been changed materially. The course of treatment consists of the injection of 0.15 gm. of stilbamidine once daily for 14 days. The therapeutic pattern of stilbamidine in tic douloureux appears to be consistent in a significant number of patient. The complete relief of pain for periods longer than the usual remission in the cycle of tic douloureux has been seen repeatedly. The late effect or delayed neuropathy, appearing long after the administration of stilbamidine, makes a possible psychogenic therapeutic effect less likely. In contradistinction, patients with tic douloureux usually experience a severe exacerbation of their pains during the first weeks of waiting for the delayed neuropathy. Unquestionably, in a disease such as tic douloureux wide latitude in choice of treatment will exist until the cause of the disease is determined. More specific therapy may then be used. A statistical analysis of the results obtained in the treatment of tic douloureux with stilbamidine compares favorably with other methods. Stilbamidine, unlike sterile water, is neurotoxic, and relief can be obtained in the treatment of tic douloureux with stilbamidine.

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Veterans Administration Hospital
Fort Howard, Md.

* Correspondence, J.A.M.A., April 21, 1956, p. 1431.

Book Reviews

Clinical Laboratory Diagnosis, by Samuel A. Levinson, M.D., and Robert P. MacFate, Ph.D., Lea & Febiger, Philadelphia, 1956.

This new edition by two masters in the field brings current advances in methodology up-to-date even though the application of the newer procedures in clinic and hospital wards necessitates considerable experience before they can be considered applicable. Each chapter represents a complete survey of the subject with ample references to the current literature. Data are presented clearly to enable a rapid review of the pertinent facts on the subject correlating basic sciences with clinical interpretations. Newer techniques are given for examinating biologic materials. Pathologic findings are compared with the normal and correlated with clinical pathology. The book is unique in that each phase of the body subject to study is correlated with laboratory procedures for determining the functional status. Special emphasis is given to the mouth invaluable to the dentist in recognizing and quantitating pathological conditions. This is followed by a consideration of other portions of the gastrointestinal tract, metabolic evaluations, blood studies, renal function, hematological procedures and advances in immunology and serology, bacteriology, analysis of milk and water, tropical diseases and even toxicology. The book is a revelation of current procedures and their interpretations applicable to everyday problems. It enables the dentist to perceive the role of laboratory studies in systemic disorders as they relate to affections of the mouth.

I. NEWTON KUGELMASS, M.D.

Oral Cancer and Tumors of the Jaws, By George S. Sharp, M.D., F.A.C.S., F.A.C.R., Weldon K. Bullock, M.D., M.Sc., and John W. Hazlet, D.D.S. The Blakiston Division, McGraw Hill Book Company, Inc., New York, 1956, $15.00.

The dentist as an oral physician is the modern need. This need is being met by undergraduate and postgraduate courses in their emphasis of oral medicine. New schools of dentistry, not bound by precedent or by well established departments are building their curricula around oral medicine as the hub with other branches such as restorative phases placed in a more minor perspective. A good example is the new dental school in Israel.

In order to permit such curricular changes, the appropriate textbooks must be made available. As with many subjects at their early stages of development, several inferior books have appeared on the market on the subject of oral cancer which, because they had no standard for comparison, had fairly wide distribution.

It is pleasing to find that this subject at last has attained its maturity in texts of the type of the one under review. It is well written, well organized, well illustrated and extremely instructive. The dentist or physician reading this volume can become aware of the differential diagnosis of the many types of benign and malignant lesions of the oral cavity and its adnexa. This book has much new material and presents it lucidly. The reader may be surprised to learn that 25% of all oral malignances appear on the gingivae. He will understand with ease the technique and indication for biopsy. He will see many examples of the various benign and malignant changes revealed in photographs and roentgenographic representations. Choice of treatment is given adequate coverage.

All in all, we have here a book which, prepared by a combined medical-dental team, bridges still further the gap between the two professions.

SAMUEL CHARLES MILLER

Hypnosis and Its Therapeutic Appplications, by Roy M. Dorcus, Ph.D., Blakiston Division, McGraw-Hill Book Company, Inc., New York, 1956, $7.50.

This eclectic study of hypnosis is of value because it includes the theoretical, experimental and clinical aspects of this complex subject. Much of the literature in this field is either theoretical and experimental or largely clinical. The authors, seven psychologists and one dentist have incorporated their own experiences into their chapters.

The section devoted to Methods of Induction is a fine one. It is emphasized, however, that induction cannot be stereotyped, but must be designed to suit the needs of the individual patient.

Dr. Kirkner has written a revealing treatise on the Control of Sensory and Perceptive Functions by Hypnosis, substantiated by numerous charts and tables.

In the chapter on the use of hypnosis as a Diagnostic Tool, Dr. Dorcus demonstrates that "in many instances diagnosis becomes interwoven with therapy and may actually depend on the outcome of the therapy. If it is demonstrated that suggestive therapy eliminates or ameliorates the symptoms, one is on reasonably safe ground in assigning their origin to psychogenic causes. To illustrate, he cites numerous cases.

The sections on Hypnosis in General Hospital Service and Hypnosis in Dentistry will doubtless interest many in those professions.

The authors view hypnosis as a tool in diagnosis of treatment not as a panacea. It is up to the individual practitioner to determine when and how suggestive therapy can be of value to him.

Clinical Hematology, by Maxwell M. Wintrobe, M.D., Ph.D., Fourth Edition, Lea & Febiger, Philadelphia, 1956.

This Fouth Edition crystallizes the current concepts on disorders of the blood featuring the recent advances in every phase of the field especially abnormal hemoglobin syndromes, blood groups, blood transfusion production and destruction of red corpuscles, blood clotting mechanism, hemorrhagic disorders and hemolytic anemias. The work is not only an interpretation of the clinical features of blood in health and in disease but an evaluation of histology, physiology, biochemistry and pathology as each applies to disease at the bedside. Emphasis throughout the book is on diagnosis and treatment of diseases of the blood in all climes and communities. The tests are presented simply and concisely to enable them to be performed by the practitioner. There is considerable new practical data, both clinical and laboratory, to unravel the oral preludes of blood diseases, of systemic origin invaluable to the scholarly dental clinician. The chapters are a revelation of clarity and comprehensiveness: The Origin and Development of the Cells of the Blood in the Embryo, Infant and Adult; The Erythrocyte; The Leukocyte; Blood Platelets and Coagulation; The Blood as a Whole; Blood Groups and Blood Transfusions; The Principles and Technic of Blood Examination; Anemia; General Considerations and Treatment; Pernicious Anemia and Related Macrocytic Anemias; The Normocytic Anemias; Hemolytic Anemias; Anemia and Other Disorders in Childhood; The Purpuras; Hemophillia and Other Hemorrhagic Disorders; Leukemia; Tumors and Tumor-like Conditions Involving the Blood-Forming Organs and Agranylocytosis and Infectious Mononucleosis. There is no substitute for Wintrobe's Clinical Hematology this year.

I. NEWTON KUGELMASS, M.D.

Blakiston's New Gould Medical Dictionary, by Normand L. Hoerr, M.D. and Arthur Osol, Ph.D., Editors. With the cooperation of an editorial board and 88 contributors. Ed. 2, The Blakiston Division, McGraw-Hill Book Co. Inc. New York, 252 illustrations on 45 plates, 129 in color, 1956, $11.50.

This new volume is a fully revised up to the minute medical and dental dictionary and is the work of a distinguished editorial board and 88 outstanding contributors from specialized fields. The book contains 12,000 new terms, 8,000 changes with spellings and usages modernized throughout. The new edition keeps abreast of the very latest developments in medicine, dentistry and allied fields. Drugs in various stages of animal and clinical investigation have been defined. The editors have omitted obsolete or unused terms in order to present a dictionary that is practical to the general practitioner.

The appendix to this valuable dictionary contains tables covering anatomic considerations, blood constituents, elements, enzymes, hormones, isotopes, medical signs and symbols, prescription writing, proteins, vitamins, weights and measures, etc. This information, appearing in table form, will be of great aid to the general practitioners of medicine and dentistry.

This illustrated medical and dental dictionary should be in the library of every physician and dentist.

Complete Mouth Rehabilitation Through Crown and Bridge Prosthodontics, by Harry Kazis, D.M.D., and Albert J. Kazis, D.M.D., 392 pages, 551 illustrations on 332 figures, Lea and Febiger, Philadelphia, 1956, $15.00.

This text is written primarily to acquaint the student with the possibilities of complete mouth rehabilitation and to give the dentist a helpful guide in the diagnosis, planning and treatment of these complex cases.

The material presented is considered under three main divisions. The fundamental theories and basic principles of mouth rehabilitation are considered first. It is emphasized that in order to render a true health service one must have a fundamental knowledge of the anatomical structures and an understanding of the physiological mechanism of the mouth and its related components.

Chapters on physiologic rest position and occlusion precede an evaluation of the periodontal considerations in crown and bridge prosthodontics.

There are numerous case histories illustrating the techniques of treatment employed by the authors. Included are discussions of the operative and technical phases of rehabilitation as well as a photographic description of the fabrication of the precision attachment partial denture. The authors have undoubtedly added a valuable contribution to the field of oral rehabilitation.

MARVIN N. OKUN

A Textbook of Operative Dentistry, by William H. O. McGehee, D.D.S., M.D., F.A.C.D., Harry A. True, D.D.S., F.A.C.D., and E. Frank Inskipp B.S., D.D.S., F.A.C.D. Blakiston Division, McGraw-Hill Book Company, 720 pages, 423 illustrations, 1956, $14.00.

The fourth edition of Operative Dentistry contains concise and detailed procedures for tooth restorations. Included is an analysis of the relative values of several restorative materials as well as a classification of their qualities.

"Accuracy with speed" is a new chapter devoted to a technique whereby the operator can decrease the chair time necessary for various operative procedures. A complete description of the vacuum method for the removal of all water and cuttings as rapidly as

they accumulate is also included.

A very comprehensive chapter devoted to operative dentistry for children is one of the highlights of the text. In it are discussed patient handling, roentgenographic examination and other perplexing problems encountered in the child patient.

Many fine illustrations are used to clarify the procedures presented. This book can be recommended as a worthwhile addition to the dental library.

A Manual of Oral Surgery by W. Harry Archer, B.A., M.A., D.D.S., ed. 2, W. B. Saunders Company, Philadelphia, 877 pages, 1400 figures, 1956, $16.50.

With the assistance of eleven contributors, Dr. Archer's second edition is an excellent up to date volume on oral surgery covering all phases of this vast subject. This text is not only a manual for those interested in oral surgery, but it can serve as an excellent reference volume for those interested in dental medicine, prosthesis and oral pathology.

The author and publisher are to be congratulated on the fine illustrations, diagrams and drawings that make up this manual. The roentgenographs of the teeth, supporting structures and the skull are clear and illustrate the points under discussion.

Dr. Harry Sicher has contributed a chapter on "Anatomic Explanation of Pain in and About the Oral Cavity." An interesting chapter on "Oral Radiographic Localization" was written by Dr. J. C. Eselman, and Dr. Hamilton B. G. Robinson has written about "Oral Malignancies: The Dentist's Responsibility." Several other contributors have written outstanding chapters which enhances the value of this book to both the general practitioner and the oral surgeon.

This reviewer, who studied under the author in his undergraduate days, well remembers the proficiency and enthusiasm for oral surgery that Dr. Archer imparted to all his students. His keen perception of this subject is manifested throughout the pages of this text. This step-by-step atlas of operative techniques is a must for every practitioner.

IRVING YUDKOFF

Periodontal Therapy by Henry M. Goldman, D.M.D., F.A.C.D., Saul Schluger, D.D.S., and Lewis Fox, D.D.S., F.A.C.D., C. V. Mosby Company, St. Louis, 190 text illustrations and 100 plates, 565 pages, 1956, $18.75.

This new text about periodontal therapy is actually divided into two basic parts; one containing the description, explanation and rationalization of techniques and results, and the other is an atlas, self-contained, in the plates and their explanatory legends. The drawings are for the most part of actual clinical situations. In some cases drawings were used to supply the step-by-step method between preoperative and postoperative pictures.

The three authors are primarily teachers in graduate and undergraduate periodontics and direct this text at the practitioner who is specializing or intends to specialize in periodontia. The general practitioner can use this book as a ready reference and can easily follow the step-by-step procedures. All standard methods of periodontal treatment have been described and illustrated. In addition, much space has been devoted to the variation from the standard.

This is a welcome addition to the group of many fine texts on periodontal therapy.

Academy Proceedings

AMERICAN ACADEMY OF DENTAL MEDICINE ANNOUNCES MID-ANNUAL MEETING

The American Academy of Dental Medicine will hold its Eleventh Mid-annual Meeting and Luncheon at the Hotel New Yorker, New York City, on Sunday, December 2, 1956. There will be a business meeting for members at 10 A.M. This will be followed by a luncheon and scientific sessions for Academy members and friends starting at 12:30 P.M.

The afternoon scientific session will be devoted to a symposium on "Recent Concepts on the Temporomandibular Joints in Relation to Fixed and Removable Dentures and Roentgenographic Interpretation."

Participating in this symposium will be:

1. Dr. Morton Amsterdam, Philadelphia, Pa., who will discuss the "Interrelationship of the Temporomandibular Joints and Occlusion in Fixed Prosthesis."
2. Dr. Lewis S. Block, Louisville, Ky., will speak on "The Prosthodontist and the Temporomandibular Joint Syndrome."
3. Dr. William J. Updergrave, Philadelphia, Pa., will cover the "Radiographic Analysis of Functioning Temporomandibular Joints."
4. Dr. Samuel Charles Miller, New York, N.Y., will discuss and summarize these three presentations.

All members and interested dentists and physicians are cordially invited to attend. For reservations and programs address the Chairman, Dr. Abraham Reiner, 114-01 201st St., St. Albans 12, New York, or Dr. Andrew Cannistraci, 2152 Muliner Avenue, The Bronx, New York.

Eleventh Mid-Annual Meeting Committee
Dr. Abraham Reiner, *Chairman;* Dr. Alonzo DeVanna, *Co-Chairman;* Dr. Andrew Cannistraci, Dr. George Goldberg, Dr. Sanford Kirsch, Dr. Stephan Nachbar.

TAPE RECORDINGS AVAILABLE OF TENTH ANNUAL MEETING'S SPEAKERS

Duplicate tape recordings of the essayists' presentations before the Tenth Annual Meeting of the American Academy of Dental Medicine are being made available to the Academy membership by the Michigan Section. The following tapes can be secured from Dr. Herman M. Lifton, 2920 Oakwood Blvd., Melvindale, Michigan.

1. "Oral Manifestations of Endocrine and Nutritional Imbalances," Medical Aspects by Dr. Lawrence Alexander and Dental Aspects by Dr. Donald Kerr. Running time, 2 hours, $5.00.

2. "Oral Manifestations of Circulatory Disturbances" by Dr. Raymond Monto, and "Oral Dermatologic and Allergic Manifestations of Systemic Disease" by Dr. Alice Palmer. Running time, 3 hours, $6.00.

3. "Oral Radiologic Manifestations of Systemic Disease" by Dr. Edward C. Stafne, and "Laboratory Tests of Aid in Oral Diagnosis" by Dr. William Umiker. Running time, 3 hours, $6.00.

4. "On Dental Writing" by Dr. Jacques P. Gray. Probable cost, $4.50.

NEW MEMBERS ELECTED AT TENTH ANNUAL MEETING OF AMERICAN ACADEMY OF DENTAL MEDICINE — JUNE 23, 1956

BAILEY, NORMAN C.
Fredericksburg, Va.

BELJAN, MATTHEW, R.
Cleveland, Ohio

CAMPBELL, ULYSSES
East Orange, N. J.

CHESUAY, GASTON
Montreal, Canada

CHILTON, NEAL W.
Trenton, N. J.

CLARK, RICHARD J.
Appleton, Wisconsin

COWAN, MARTIN M.
Detroit, Mich.

DIONNE, PAUL
Joliette, Canada

ENGELMAN, MELVIN A.
Wappingers Falls, N. Y.

ENTINE, MARTIN
Philadelphia, Pa.

ESKOW, GERALD V.
Baltimore, Md.

FRANKEL, SHELDON
Toledo, Ohio

GALLAGHER, JOHN F.
Milton, Mass.

GAYNOR, SOLL
Flint, Michigan

GENN, BENJAMIN H.
Pittsfield, Mass.

GLASSER, MORRIS M.
Boston, Mass.

GRINNELL, HERBERT W.
Brooklyn, N. Y.

GROSSMAN, MAURICE F.
Lawrence, Mass.

HERSCHFUS, LEON
Detroit, Michigan

JACOBSON, FREDERIC L.
Seattle, Wash.

KANDERS, RALPH F.
Montclair, N. J.

KANEV, PHILLIP S.
Philadelphia, Pa.

KENNEDY, RICHARD W.
Philadelphia, Pa.

KRUTCHIK, DAVID
Cheektowaga, N. Y.

KUHN, LAWRENCE M.
Bellerose, L. I.

LECO, EUGENE
No. Attleboro, Mass.

LEHRER, LOUIS
New York, N. Y.

LIBASSI, JOSEPH P.
Brooklyn, N. Y.

MARCUS, MORTON
New York, N. Y.

MAYNE, JAMES L.
Beverly, Mass.

NISBET, THOMAS G.
Charlotte, N. C.

ORLEAN, SOL L.
Charlotte, N. C.

PEERY, WALTON S.
Charlotte, N. C.

POHLE, ISHMAEL T.
Grosse Pointe Woods, Mich.

POOR, WILLARD H.
Boston, Mass.

POWELL, HUME S.
Richmond, Va.

PRASERTSUNTARASAI, T.
Birmingham, Ala.

PUCCIO, JOSEPH F.
Port Washington, N. Y.

RABBIN, MICHAEL
Phoenixville, Pa.

ROBBINS, ALLAN H.
Cleveland, Ohio

ROBERTS, PEARCE, JR.
Asheville, N. C.

RUEGER, ARVON E.
Plainville, Kan.

SANDLER, HENRY
Brooklyn, N. Y.

SCHAFFER, JACOB
Newark, N. J.

SCHIED, EDWARD L.
Jackson, Mich.

SEDELL, LEONARD A.
Jersey City, N. J.

SHERMAN, CHARLES W.
College Park, Ga.

SHKLAR, GERALD
Boston, Mass.

SIDAHMED, SHAFIK
Alexandria, Egypt

SKWAREK, LEONARD R.
East Detroit, Mich.

SNIDERMAN, MARVIN
Pittsburgh, Pa.

WEBB, WILLIAM C. JR.
Dallas, Texas

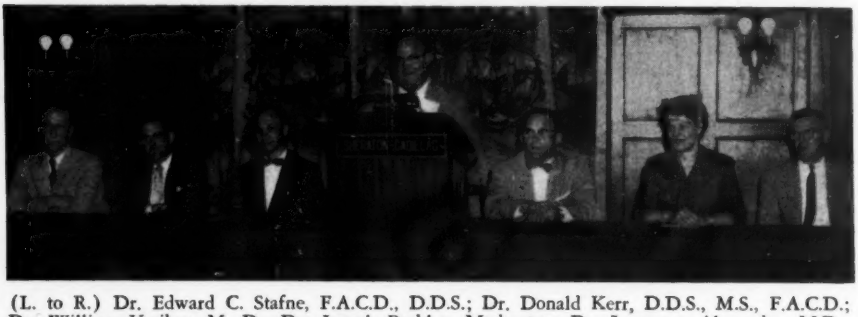
WERTHEIMER, FRED. W.
Lansing, Mich.

WOHLFELD, STANLEY H.
Cheektowaga, N. Y.

COMMITTEE ON EDUCATION SPONSORS BIOPSY CLINIC

On Monday morning, December 3, 1956 the Committee on Education of the Academy will sponsor a "Biopsy Clinic" to be held at the Veterans Administration Hospital, 24th Street and First Avenue, New York, N. Y. This clinic is being arranged through the courtesy of Dr. I. W. Scopp, Chief of Dental Service at the hospital. Attendance will be limited. Please contact Dr. Harold E. Brennan, Chairman, 400 Madison Avenue, New York 17, N. Y. for a reservation to attend the "Biopsy Clinic."

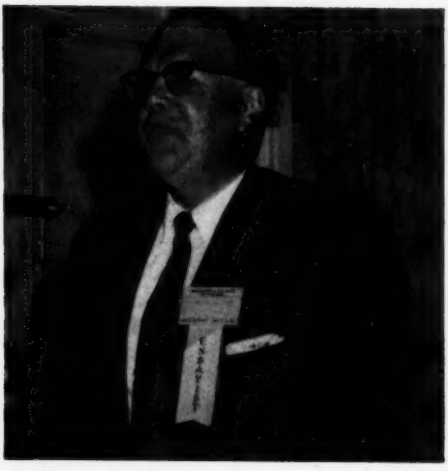
ESSAYISTS — TENTH ANNUAL MEETING



(L. to R.) Dr. Edward C. Stafne, F.A.C.D., D.D.S.; Dr. Donald Kerr, D.D.S., M.S., F.A.C.D.; Dr. William Umiker, M. D.; Dr. Joseph Barkley, Moderator; Dr. Lawrence Alexander, M.D.; Dr. Alice Palmer, M.S., M.D.; and Dr. Raymond Monto, M.D., F.A.C.P.

The above group of essayists presented excellent papers before the Tenth Annual Meeting of the American Academy of Dental Medicine which was held in the Sheraton-Cadillac Hotel in Detroit, Michigan, June 21 through 24, 1956. Examples of these fine papers can be found in this issue of the Journal which contains Doctors Palmer and Kerr's articles.

LUNCHEON SPEAKER



Dr. Jacques P. Gray, Director of Special Medical Services of Parke Davis and Company and visiting lecturer on medical writing, under the auspices of the Education Committee of the American Medical Writers Association, delivered an informal talk entitled "On Dental Writing" before the Academy. His humorous approach to this important topic was very well received.

10TH ANNUAL CONVENTION CHAIRMAN

Dr. James R. Ladd, dynamic Chairman of the Tenth Annual Convention of the American Academy of Dental Medicine. Together with his Co-Chairman, Dr. D. Chalmers Lyons and their committee, they ran one of the best Academy Conventions since its inception.

Announcements

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE

NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The following postgraduate courses in Periodontia and Oral Medicine will be offered at the New York University College of Dentistry and will be given by Dr. Samuel Charles Miller and Staff:

No. 124. REFRESHER COURSE IN PERIODONTIA AND ORAL MEDICINE
Ten sessions, Wednesdays, 4:00 to 6:00 P.M.
February 20, 1957 to May 1, 1957.
Tuition and books. $156.00. Instruments approximately $55.00.

Theoretical and practical application of periodontal diagnosis and techniques to general dentistry. Four lecture and six clinical demonstration sessions. Equilibration of occlusion is performed by the student on casts. Patient demonstrations include equilibration of occlusion, conservative and radical therapy, home care, etc.

No. 123. PERIODONTIA AND ORAL MEDICINE IN THE SPANISH LANGUAGE
Four weeks, Monday through Friday, 9:00 A.M. to 4:00 P.M.
March 18 to April 12, 1957 inclusive.
Tuition including books and instruments approximately $385.00.

Theoretical and practical application of Periodontia and Oral Medicine is fully explained and demonstrated. Equilibration of occlusion is performed by the student on casts. Lectures and clinical demonstrations of diagnosis and treatment planning, conservative and radical techniques, equilibration of occlusion, home care, etc. The entire course is conducted in the Spanish language.

No. 122. COMPREHENSIVE PREPARATION IN PERIODONTIA AND ORAL MEDICINE

No. 122 A. One academic year, full time. September 17, 1956 to May 24, 1957.
Tuition $1,010.00. Instruments and books approximately $135.00.

No. 122 B. Two academic years, full time. September 17, 1956 to May 23, 1958.
Tuition $1,770.00 (two years). Instruments and books approximately $231.00.

No. 122 C. Two academic years, half time. September 17, 1956 to May 23, 1958. (Two and one half days weekly.)
Tuition $1,010.00 (two years). Instruments and books approximately $135.00.

No. 122 D. Four academic years, half time. September 17, 1956 to May 27, 1960. (Two and one half days weekly.)
Tuition $1,770.00 (four years). Instruments and books approximately $231.00.

These courses are designed for comprehensive preparation in Periodontia and Oral Medicine and are of particular significance for certification towards specialization. All didactic and clinical phases are covered with the objective of developing expert proficiency.

TUFTS UNIVERSITY SCHOOL OF DENTAL MEDICINE

The following courses listed below will be presented at Tufts University School of Dental Medicine during 1956-1957. Application and further information may be obtained by writing the Director, Division of Graduate and Postgraduate Studies, Tufts Univeristy School of Dental Medicine, 136 Harrison Avenue, Boston 11, Mass.

DPG. 102—Clinical Endodontics. Monday through Friday, March 25-29, 1957. Tuition $150.00—Class limited to twelve. Dr. Ralph F. Sommer, Professor of Dentistry, University of Michigan.

DPG. 302—General Anesthesia. Each Wednesday for Eight Weeks from Jan. 9 to Feb. 27, 1957. Tuition $150.00—Class limited to fifteen. Dr. Edward Sleeper and Staff.

DPG. 401—Orthodontic principles of Interest to the General Practitioner. Each Wednesday for Eight Weeks from Feb. 6 to March 27, 1957. Tuition $150.00—Class limited to ten. Dr. Herbert I. Margolis and Staff.

DPG. 402—Applied Cephalographics for Orthodontists. Wednesday, Thursday, Friday, April 24, 25, 26, 1957. Tuition $100.00—Class limited to ten. Dr. Herbert I. Margolis and Staff.

DPG. 501—Clinical Dentistry For Children. Each Friday for Six Weeks from Oct. 19 to Nov. 23, 1956. Tuition $150.00—Class limited to ten. Dr. Frederic Shiere and Dr. Helmi Fogels.

DPG. 502—Clinical Dentistry For Children. Tuesday, Wednesday, and Thursday, June 18, 19, 20, 1957. Tuition $75.00—Class limited to 12. Dr. Maury Massler, Professor of Pedodontia, University of Illinois, School of Dentistry.

DPG. 702—Crown and Bridge Prosthesis—Participation Course. For 6 days —March 6, 7, 13, 14, 20, 27, 1957. Tuition $200.00—Class limited to 8. Dr. R. Sheldon Stein, Boston, Massachusetts.

DPG. 801—Occlusal Equilibration In Children and Young Adults. Wednesday, Thursday and Friday, June 5, 6, 7, 1957. Tuition $100.00—Class limited to twelve. Dr. Edward T. Fischer and Dr. Herbert I Margolis with Guest Clinician Dr. Clyde H. Schuyler.

DPG. 804—The Diagnosis and Treatment of Temporomandibular Joint Disturbances. Wednesday, Thursday, Friday, April 3, 4, 5, 1957. Tuition $100.00 —Class limited to fifteen. Dr. Sidney E. Riesner, New York City.

DENTAL GUIDANCE COUNCIL FOR CEREBRAL PALSY

The Annual Scientific Meeting of the Dental Guidance Council for Cerebral Palsy will be held in the Hartford Room of the Hotel Statler, 32nd Street and Seventh Avenue, New York City on Sunday, November 4, 1956 at 10 A.M.

The program should be of particular interest to the dental profession and all members and their friends are cordially invited to attend. Further information may be secured at the offices of the Dental Guidance Council for Cerebral Palsy, 47 West 57th Street, New York 19, N. Y., PLaza 3-3005.

GREATER NEW YORK DENTAL MEETING

New dental educational techniques, including the first long-distance transmission of closed-circuit color television clinics direct from Walter Reed Army Hospital at Washington, D. C., will feature the 32nd annual Greater New York Dental Meeting to be held at Hotel Statler, New York City, December 3 to 7, it has been announced by Dr. Roland E. Morse, General Chairman.

Another innovation to be introduced at the meeting will be a series of six Vu-well clinics, employing equipment which results in a hitherto impossible clarity and definition of projected picture.

The color television clinic program to be transmitted from Walter Reed Army Hospital, and directed by Dr. Paul Schafer, Executive Director of its Television Division, will be presented Thursday afternoon, December 6. It will include a gingivectomy performed by Col. Alfred E. Toye, Chief of the Periodontia Section of that hospital; the preparation of an anterior crown by Lt. Col. Russell H. Augsburger, Chief of the Restorative Dentistry Section; and the removal of a cyst by Col. Robert B. Shira, Director of the Division of Dentistry at Walter Reed Army Institute of Dental Research. The program has been arranged through the courtesy of Brig. Gen. Arthur L. Irons, Director of Dental Activities at Walter Reed Army Medical Center.

Projected table clinics in color will again be offered by prominent practitioners of New York and other areas. In addition, there will be the usual wide range of Registered Clinics, as well as several audience-participation discussions conducted by eminent educators and specialists.

A number of scientific exhibits depicting new research investigations will be shown as meeting adjuncts. Several hundred commercial exhibits will demonstrate the latest in dental materials and equipment.

Further information will be furnished promptly on request to Mrs. Mabel Purdy, Executive Secretary, Room 106A, Hotel Statler, New York 1, N. Y.

TEMPLE UNIVERSITY SCHOOL OF DENTISTRY POST GRADUATE COURSES — 1956-1957

Course 1. Periodontics—
Dr. Jacoby Rothner and associates, November 5 to November 17 inclusive.
Course 2. Full Denture Prosthesis—
Dr. Bernard Jankelson, November 26 to November 30 inclusive.
Course 3. Occlusion and What To Do About It—
Dr. Bernard Jankelson, December 3 to December 6 inclusive.
Course 4. Advanced Orthodontics—
Dr. Robert H. W. Strang, January 20 to February 2 inclusive.
Coure 5. Practice Administration—
Dr. Jay H. Eshleman, January 30 to February 1 inclusive.
Course 6. Periodontal Prosthesis—
Dr. Morton Amsterdam and Dr. Walter Cohen, February 11 to February 16 inclusive.
Course 7. Orthodontics in Periodontal Therapy—
Dr. Jack Alloy and Dr. Michael Fogelman, April 11 to April 13 inclusive.

For additional information and application, write to Dr. Louis Herman, Postgraduate Division, Temple University School of Dentistry, 3223 North Broad Street, Philadelphia 40, Pennsylvania.

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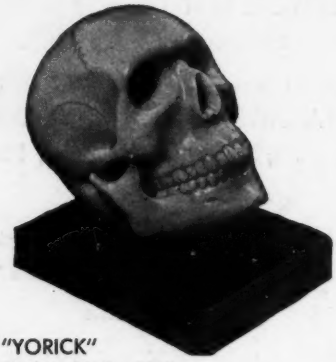

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